ARCHIVES OF

NEUROLOGY AND PSYCHIATRY

VOLUME 23
1930

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

VOLUME 23 NUMBER 1

ARCHIVES OF NEUROLOGY AND PSYCHIATRY

JANUARY, 1930

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, Jan. 7, 1919, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized Jan. 15, 1919.

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THE RÔLE OF THE HYPOTHALAMUS AND MESENCEPHALON IN LOCOMOTION*

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CHICAGO

The reflex nature of progression has been the subject of a large number of investigations, and it is generally admitted that the thalamic cat and the midbrain rabbit exhibit fairly normal progression. Progression, as exhibited by the thalamic cat, consists of at least three components, which must be properly synthesized and coordinated for walking to occur: (1) rhythmicity, which is known to be present, for the hind limbs, at least, in spinal animals; (2) static or postural tonus, which is present, according to some observers, in the spinal animal and which is much more pronounced in the decerebrate animal with transection at the level of the posterior colliculi, and (3) equilibration and maintenance of upright posture, for which the rostral portion of the tegmentum of the mesencephalon or its continuation into the hypothalamus is necessary. The decerebrate animal prepared by a transection through the mesencephalon possesses the first two of these components, but those who have worked with these animals are cognizant of the fact that such animals are unable to maintain their equilibrium and upright posture. It should be emphasized that there is a differentiation which must be made between the coordinated movements of progression and the complete act of walking. The decerebrate animal under the proper conditions of tonus distribution may show coordinated movements of progression, but no one has ever shown that an animal decerebrated by transection through the mesencephalon can walk. It might be argued that the reason the decerebrate animal does not walk is that the increased tonicity and rigidity make it impossible for the movements of progression to occur. However, there is an additional reason, in that the decerebrate animal is unable to right itself and to maintain its equilibrium.

* Submitted for publication, May 1, 1929.

* From the Institute of Neurology, Northwestern University Medical School.

* Read at the Fifty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 27, 1929.

In any consideration of experimental work of this type, it should be emphasized that the levels of section should be controlled not only grossly but microscopically. Decorticate animals are ones in which the cerebral cortex has been removed without any particular damage to the thalamus and basal ganglia. Thalamic animals are prepared by sections which remove the cerebral cortex and varying amounts of the corpus striatum and thalamus. In a hypothalamic animal, the section passes from the rostral border of the superior colliculus to the region of the optic chiasm. A midbrain animal should be one in which has been removed not only the cerebral cortex and the thalamus but also the hypothalamus. In the consideration of the literature, we shall endeavor to make the levels of the sections and the microscopic observations clear in all cases in which the information is available.

LITERATURE

Goltz [1] first removed the left cerebral hemisphere in a dog in two separate operations and afterward the right one. The basal part of each temporal lobe was allowed to remain so that the optic tracts would not be injured. Three days after the last operation, the animal was able to walk with good coordination, but it was extremely restless. Holmes [2] examined the brain of this dog and reported (p. 2):

> A trace of the cortex belonging to the olfactory area remained on the base of the brain, ventral to the head of the nucleus caudatus. The greater part of the left nucleus lenticularis was removed with the hemispheres; most of the right, on the other hand, remained. The uncinate gyri of the temporal lobes, which were purposely left behind, were the solitary representatives of the hemispheres, but the right was shrunken and degenerated, and the left consisted of merely a thin-walled cyst containing clear fluid.

Dusser de Barenne [3] extirpated a considerable portion of the cerebral hemispheres in two cats and succeeded in keeping them alive for several months. The extent of the decortication was greater in the second than in the first, but even in the second, according to Brouwer,[4] a considerable portion of the archipallium was present. In cat 1, damage was done to the caudatum and putamen, and there was some pathologic change in the globus pallidus. In cat 2, nearly all of the caudatum and putamen was removed on both sides. Dusser de Barenne said that cat 2 is to be compared with the dogs of Rothmann and Goltz from the physiologic point of view. Neither of these cats exhibited decerebrate rigidity and both showed the righting reflexes shortly after the operation. They were able to walk in fairly normal posture, but during the first few days exhibited some abnormality, especially in the

1. Goltz, F.: Arch. f. d. ges. Physiol. **51**:570, 1892.
2. Holmes, G. M.: J. Physiol. **27**:1, 1901.
3. Dusser de Barenne, J. G.: Arch. néerl. de physiol. **4**:31, 1920.
4. Brouwer, B.: Arch. néerl. de physiol. **4**:124, 1920.

fore limbs, which consisted of walking on the dorsums of the forefeet. After a few days, this disappeared so that only rarely the dorsum of the foot touched the ground.

Rothmann [5] gave a complete description of a decorticated dog which his father had prepared and which lived for more than three years. He said (p. 307):

> Das Lagegefühl war bei unserem Hunde, wie der Versenkungsversuch zeigte—die Beine hingen dabei in spastischer Streckstellung herab—gestört. Doch war das Lagegefühl keineswegs aufgehoben, und später lernte der Hund richtig und schnell Lagegefühlsstörungen zu korrigieren.

This dog improved as time went on so that he could perform complex reflexes, but we cannot give any description of these reactions here. The thing that interests us is that the animal could walk and run about with a fairly normal distribution of tonus in the musculature. The neopallium was completely removed, but some portions of the archipallium and paleopallium remained. There is some question whether the last two were functional. The nucleus caudatus and putamen were primarily injured in the operation, and it is difficult from the description to determine how extensive the injury was, a point which it would be necessary to know before making any functional distribution to these parts as has been done. The globus pallidus was not injured by the operation. The absence of any marked degeneration in the substantia nigra is reported.

Dresel [6] removed the cerebral hemispheres and the neostriatum on both sides in a dog, and he said that the globus pallidus was completely removed. However, if the globus pallidus is situated in the dog as Winkler and Potter [7] show it to be in the cat, this operation would have left the portion of the pallidum between the cerebral peduncle and the optic tract intact. The operation was done in two stages, and the animal lived for three months after the second stage. Two days after the second operation, the animal could stand and move about. It moved about only when stimulated externally or internally. It was restless when the bladder was distended and when hungry; at such times it ran about with a depressed head. The animal lacked spontaneity and showed no tendency to develop complex reflexes. The author believed that the failure to acquire more complex reflexes, as the Rothmann dog did, was due to the removal of the corpus striatum, which is said to have taken over, to some extent, the function of the cortex in the Rothmann dog. It should be remembered in this connection that the Goltz dog showed spontaneity and some ability to combine simple

5. Rothmann, H.: Ztschr. f. d. ges. Neurol. u. Psychiat. **87**:247, 1923.
6. Dresel, K.: Klin. Wchnschr. **2**:2231, 1924.
7. Winkler, C., and Potter, A.: An Anatomical Guide to Experimental Researches on the Cat's Brain, Amsterdam, W. Versluys, 1914.

movements into more complex reactions after nearly complete removal of one lenticular nucleus. Furthermore, in the Rothmann dog, the globus pallidus alone in the lenticular nucleus was uninjured. The interesting thing to us is that, with nearly complete removal of the corpus striatum and globus pallidus, the dog was able to walk and maintain an upright posture with a "normal" distribution of tonus. However, the question still remains whether or not extensor hypertonus might not have appeared in some postures, if means had been taken to show it.

In all these experiments, with removal of most of the cerebral cortex, with extirpation of varying amounts of the neostriatum and paleostriatum and with varying injuries to the thalamus, the animals were able to maintain an upright posture, walk and exhibit a fairly normal distribution of tonus. From the work of Dresel [6] it seems evident that all the neostriatum and nearly all the paleostriatum can be removed without interference with the walking pattern or any marked disturbance in the mechanism regulating tonus.

The importance of the cerebral cortex gradually increases as one passes from lower to higher mammals, and one should be exceedingly careful in transferring observations made on an animal such as the rat to man. However, observations made on the lower mammals give an index to the progressive functional importance of various parts of the central nervous system. In the Virginian opossum (a marsupial), the cerebral hemispheres are of a primitive type. Rogers [8] observed that extirpation of the excitable cortex without injury of the corpus striatum was followed by temporary weakness of the contralateral fore leg and difficulty in the use of the hand in climbing and grasping. This persisted only a few days and gradually passed away. His experiments show that the corpus striatum in the opossum exerts a motor or tonic action on the musculature of the anterior part of the body. His opossum 32 is the most interesting of his animals, for here not only the cerebral cortex (with exception of a few olfactory connections) but also the corpus striatum was completely removed on both sides. This animal walked about the room and showed decerebrate restlessness. Later, this restlessness was replaced by periodic activity with intervening periods of sleep. These movements were found to be inhibited by feeding and defecation, and the author believed that the walking movements were initiated by hunger or gastro-intestinal stimuli, and that they ceased following the removal of these stimuli. Lashley [9] removed various parts of the cerebral cortex in the rat and suggested that the primary function of the cerebral motor structures in the rat is the

8. Rogers, F. T.: J. Comp. Neurol. **37**:265, 1924.
9. Lashley, K. S.: Brain **44**:255, 1921.

regulation of postural reflexes. Herrick [10] pointed out, however (p. 165): "that when all the available evidence is reviewed, it is clear that in marsupials, rodents, and other lower mammals cortical differentiation is still in its incipiency."

In cats, Warner and Olmsted [11] observed that if they removed one cerebral hemisphere or one frontal area, there was an increase in tonus on both sides of the body, but the effect was predominantly contralateral. They traced the inhibitory pathway through the mesial part of the internal capsule to the pons, and then into the cerebellum by way of the middle cerebellar peduncle. The pathway corresponds in general with the corticopontocerebellar path. Weed [12] traced this pathway in the cat. Olmsted and Logan [13] removed various portions of the cat's cortex about the sulcus cruciatus. Removal of all the area surrounding the sulcus cruciatus on both sides resulted in temporary paralysis and pronounced extension phenomena. If only the frontal areas on both sides were destroyed, there was no paralysis but the extension phenomena were present.

When King [14] removed the electrically excitable area of the cortex in cats, there was a transient weakness which disappeared after forty-eight hours. The extirpation of the frontal cortex rostral to the electrically excitable area resulted in postural and tonic abnormalities in the contralateral extremities. Incoordination was more marked and more persistent than after removal of the electrically responsive areas. For the first few days after operation, the legs tended to shoot out into extreme extension on any attempt to walk. Later, the cats were able to walk, but were never able to perform more delicate movements such as jumping on or off a shelf. When the cats were held in abnormal positions, they made no effort to escape. When held suspended by the neck and tail, they exhibited hyperextension of both the fore leg and the hind leg on the side opposite the lesion, with resistance to passive flexion. There was no motor weakness to be observed in cats with chronic preparations, but the gait was seriously disturbed. For the first few days immediately following the operation, it was possible to demonstrate incoordination, which was progressively compensated, but which persisted throughout life. He stated:

> For the first few days posture was particularly abnormal. The fore-legs had a tendency to shoot backward into extreme extension, the hind-legs to extend in an anterior direction between the fore-legs. In walking the foot-pads were often turned under at the wrists, a fact which may be of increased significance when

10. Herrick, C. J.: Brains of Rats and Men, Chicago, University of Chicago Press, 1926.
11. Warner, W. P., and Olmsted, J. M. D.: Brain **46**:189, 1923.
12. Weed, L. H.: J. Physiol. **48**:205, 1914.
13. Olmsted, J. M. D., and Logan, H. P.: Am. J. Physiol. **73**:570, 1925.
14. King, W. T.: Am. J. Physiol. **80**:311, 1927.

it is remembered that the increased extensor tonus of decerebrate rigidity rarely affects the wrists of the cat. In walking the extremities seemed stiff and the high-stepping gait well described by Olmsted and Logan [15] was noted in several of the animals. For some days, the cats were not able to sit down due to inability to flex the legs and they were found constantly upon their feet. After a long period, they appeared to be able to handle the tonic extremities more satisfactorily.

These experiments were controlled to show that the hyperextension was not an irritation phenomenon. The extensor tonus was never transient, but persisted for months. Removal of the electrically excitable area was never followed by hypertonus. King concluded that the extensor hypertonus was a release phenomenon following removal of a specific area of the cerebral cortex which normally appears to inhibit extensor tonus in the crossed extremities. He interpreted the discrepancy which exists between his results and those of Magnus and Rademaker as follows:

> It may be that this spontaneous walking, which persists in cats as long as structures in the region of the posterior two-thirds of the thalamus are intact (Laughton,[15] 1924), constitutes an element which masks considerably the subsequent postural phenomena, until proper procedures are taken to demonstrate them.

This explanation seems plausible in the light of some of the observations that we shall describe later.

In a like manner, Langworthy [16] found that removal of the electrically excitable area of the cortex in the cat caused a transient paralysis from which the animal recovered. Removal of the area frontalis produced abnormal and persistent extensor hypertonus of the contralateral limbs. He found that bilateral section of the corticospinal pathway by median section of the medulla so as to sever the decussation of the pyramids was not followed by extensor hypertonus. On the sixth day, the cat could walk, but was ataxic. After this time, walking steadily improved. In nine cats, the ventral surface of the brain stem was exposed and the pontile fibers were cut in the midline. This was followed by an abnormal and increased tonus of the extensor musculature of the extremities. Emphasis is placed on the similiarity between the animals with the pontocerebellar fibers cut and those with the area frontalis removed bilaterally. This is taken to mean that the area frontalis controls the postural reflex through connections with the cerebellum.

Scott [17] was of the opinion that rigidity in the decerebrate animal may well be explained merely on the basis of the removal of cortical inhibition. A greater portion of the cortex was removed superficially

15. Laughton, N. B.: Am. J. Physiol. **70**:358, 1924.

16. Langworthy, O. R.: Bull. Johns Hopkins Hosp. **42**:20, 1928.

17. Scott, F. H.: Am. J. Physiol. **81**:507, 1927.

in dogs, cats and monkeys, and in all cases a well marked generalized muscular rigidity appeared. These observations are difficult to interpret in the light of the other experiments in decortication which have been made.

Contrary to the observations of many of the investigators just mentioned, Laughton [18] found that unilateral extirpation of the motor area in the cat and the dog results in an extensor rigidity in the contralateral legs which persists for a longer period than the paralysis following the ablation of this part of the cortex. Unilateral extirpation of the area frontalis in cats failed to produce any extensor rigidity in the contralateral limbs. The extensor hypertonus described here following removal of the motor cortex resembles rigidity in the decerebrate animal in the manner in which it is modified by mechanical stimuli and also by neck and labyrinthine reflexes, as described by Magnus and de Kleijn.[19] It differs from the rigidity of the decerebrate animal in that it does not persist undiminished for long periods of time and can be demonstrated only when the animal is quiet. The results reported by McKibben [20] confirm the observations of Laughton in his experiments with ablation.

Bernis and Spiegel [21] found that removal of the frontal pole of the cerebral cortex of the cat led to an increase of tonus, more marked in the hind limbs and of low grade and short duration. Removal of the gyrus suprasylvius posterior in the temporal lobe was followed by an extensor tonus, more marked on the contralateral side in the fore limb. They believed that extirpation of the motor area of the cortex is followed by an exaggeration of extensor tonus on the opposite side and of flexor tonus on the same side. They concluded:

> Ausser der Pyramidenbahn hat nicht nur das Stirnhirn, sondern auch der Temporallappen als Ursprungsstätte tonusregulierender Systeme Bedeutung. Doch ist der Einfluss aller dieser Systeme beim Quadrupeden (Karnivoren) noch geringgradig.

Cooper and Denny-Brown [22] were led by their observations to conclude that for a given moment the excitatory units and inhibitory units lie microscopically intermingled in the cortex and that representatives of movements in opposite directions are often closely related anatomically so that they may both be stimulated by the same stimulus. If the excitatory and inhibitory units lie so close together in the motor cortex, one might expect some release from inhibition, as well as paralysis, on its removal.

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18. Laughton, N. B.: Am. J. Physiol. **85**:78, 1928.
19. Magnus, R., and Kleijn, A. de: Arch. f. d. ges. Physiol. **145**:455, 1912.
20. McKibben, P. S.: Anat. Rec. **42**:57, 1929.
21. Bernis, W. J., and Spiegel, E. A.: Arbeiten Neurologische Institut, University of Vienna, 1925-1926, vol. 27 and 28, pp. 197-224.
22. Cooper, S., and Denny-Brown, D.: Proc. Roy. Soc., London (ser. B) **102**:222, 1927-1928.

From the standpoint of our interest here, it makes no material difference which part of the cortex needs to be removed to produce extensor rigidity, but it does concern us whether or not removal of any part of the cerebral cortex produces rigidity. We have seen that observers can remove nearly all of the cerebral cortex in animals such as the cat and the dog and still report that a normal distribution of tonus ensues. Is there an absence of extensor rigidity, or have the observers failed to examine the animals in such a manner as to bring out the presence of the extensor rigidity?

The thalamic animal differs anatomically from the decorticate animal only in that most but not all of the lentiform nucleus is removed in the thalamic animal while in the decorticate animal the latter structure is generally injured in the operation. In considering the thalamic animal, we shall include those experiments in which regions above the mesencephalon have been left intact with the brain stem. It is plain to be seen that various amounts of the thalamus would be left in these experiments. Some observers have insisted that a considerable portion of the thalamus must be functioning for the coordinated movements of progression to be present.

Thiele [23] removed the cortex from cats and monkeys, and subsequently removed the cerebral hemispheres, thalamus, mesencephalon and hind brain by successive coronal slices. He found that decerebrate rigidity did not commence till the level of section passed through the posterior part of the optic thalamus. The rigidity found with the level of section here was not so marked as that which was found when the section was made at the level of the posterior colliculus. The rigidity remained through the successive caudal sections until the region of the trapezoid body was reached. Furthermore, it was found that in no case in which the pyramidal tracts had been caused to degenerate by previous section was there any difference in the nature of the rigidity following mesencephalic section. He concluded that rigidity following decerebration is not due to a removal of a cortical influence but to removal of a thalamic one, the effect of which is transmitted along a tract that decussates high up in the mesencephalon. It is an interesting fact that he stated that rigidity can be inhibited by stimulation of the cerebral cortex as well as by stimulation of the cerebellar cortex. He stimulated the cut surface of the mesencephalon with a weak faradic current and produced coarse and slow movements of progression. His conclusion was that there is another motor tract besides the pyramidal tract, that the act of walking is not wholly dependent on the cerebral cortex, and that walking is a function which is subserved by basal centers located in the posterior part of the optic thalamus and in the mesencephalon. He thought that this other center lay in the median nucleus

23. Thiele, F. H.: J. Physiol. **32**:358, 1905.

of the thalamus and that the tract was the rubrospinal tract or one that passes down with it. He made the statement, however, that in all cases in which the mesencephalic tracts were stimulated, the posterior longitudinal bundle was stimulated.

The importance of diencephalic centers for the control of coordinated movements of progression was emphasized by Mella.[24] He performed on cats a number of ablation experiments which seemed to indicate that in the subthalamic region is an area the destruction of which does away with the automatic locomotor movements associating the fore legs and the hind legs. He was inclined to believe that the body of Luys was the center responsible for these movements. The cats did not walk with any facility and had to be supported by the nape of the neck. However, if the observations had been continued for a longer period of time, walking probably would have become more normal. Cobb said, in discussing this paper:

> One point we ought to remember; that is, that in these experiments we were interested only in the association of the progressive movements of all four legs. In spinal preparations, we may have alternate movements of one pair of legs perfectly well; and in spinal preparations you may have alternate locomotor movements during periods of stimulation. In these animals, however, the alternate movements of all four legs, in a regular rhythmic progressive reflex, were spontaneous and lasted for a long period.

The work of Magnus is summarized in his work of 1924.[25] He prepared thalamic animals in which the cerebral hemispheres and most of the lentiform nucleus were removed and in which the thalamus was mostly intact. It will be impossible to review completely the reactions. The thalamic rabbit soon after the operation maintained an upright posture with "normal" distribution of tonus and hopped in a normal fashion when stimulated. The righting reflexes were present and well developed. In the thalamic as well as in the midbrain rabbits there was a disturbance in the sense of position in the fore limbs, so that if the forepaw was placed dorsum to the ground, this abnormal posture was not corrected as quickly as in the normal animal. The thalamic cats and dogs could right themselves and could run a short time after cessation of the anesthetic and were said to have improved in from ten to fourteen days. These animals sat in a normal position, walked on stimulation and, in contrast with the rabbit, often showed "spontaneous" walking, especially if they were hungry. The righting reflexes were all present, with the exception of the optic righting reflexes. They corrected abnormal posture of the paws more slowly than normal animals would. The heat-regulating center was intact, as it was in the thalamic rabbit.

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24. Mella, H.: Diencephalic Centers Controlling Associated Locomotor Movements, Arch. Neurol. & Psychiat. **10**:141 (Aug.) 1923.

25. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924.

Magnus stated that no one had observed a shock-free thalamic monkey. He made two acute experiments (one animal lived thirty-six hours). His observations were similar to those of Karplus and Kreidl,[26] who kept one monkey alive for twenty-six days after extirpation of the cerebral hemispheres. In Magnus' thalamic experiment, the monkey could right itself and could stand if supported by one hand, but could never walk or climb. The distribution of muscle tonus was normal and decerebrate rigidity was not present. He said (p. 211):

> Da aber der Schock bei diesen Tieren bisher nicht vermieden werden konnte, ist es unbekannt, ob der Affe aus diesen Einzelleistungen, welche mit denen bei Hund, Katze und Kaninchen beobachteten übereinstimmen auch verwickeltere Bewegungskomplexe zusammensetzen kann, wie das für die genannten Tierarten geschildert wurde, und was ein Thalamusaffe tatsächlich beim Gehen, Laufen, Springen und Klettern zu leisten imstande sein wird.

He also said in regard to man:

> So sind wir doch nicht berechtigt, aus derartigen pathologischen Fällen weitgehende Schlüsse über die Maximalfunktion des Hirnstammes beim Menschen zu ziehen.

From the evidence at hand it is obvious that we cannot be sure that shock is responsible for the reactions seen in the thalamic monkey, since it may be that the cerebral cortex and the corpus striatum are essential for the appearance of the walking pattern in this animal.

Magnus prepared his midbrain rabbits by sectioning at a plane extending from the rostral border of the superior colliculus through the cerebral peduncles between the caudal border of the mammillary bodies and the point of exit of the oculomotor nerve. Rabbits thus prepared exhibited essentially the same picture as thalamic rabbits, apart from the absence of optic reflexes and the regulation of heat. The animals could right themselves, but spontaneous movements from the resting sitting position were rare, as they were in the thalamic animal. Forty-five minutes after the operation in one experiment, there was no decerebrate rigidity and the distribution of tonus in the body musculature was "normal."

The evidence presented by Rademaker[27] was interpreted by him as showing that in the cat the muscle tonus may still be normal if the brain stem is sectioned at a plane extending from the rostral border of the superior colliculi to 0.5 mm. in front of the exit of the oculomotor nerves. A close analysis, however, shows the evidence to be extremely weak. In his table, he admitted the presence of decerebrate rigidity in

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26. Karplus, J. P., and Kreidl, A.: Arch. f. Anat. u. Entwickl. physiol. Abteilung, 1914, p. 155.

27. Rademaker, G. G. J.: Die Bedeutung der roten Kerne und des übrigen Mittelhirns für Muskeltonus, Körperstellung und Labyrinthreflexe, Berlin, Julius Springer, 1926.

cats 21, 14 and 9, and said that rigidity was lacking in cat 8 and cat 18. In cat 18, the operation was completed at 10:45. From 2 to 5 o'clock in the afternoon of the same day there was, "ab und zu anfallsweise etwas erhöhter Strecktonus der Hinterbein." It is also to be noted that throughout this entire experiment the labyrinthine righting reflexes were absent. In cat 8, the labyrinthine righting reflexes were only poorly developed, and the body righting reflexes were absent throughout the experiment. The protocols indicate strongly that these animals were in poor condition, possibly still under the influence of shock, and this, rather than the presence of a center for the normal regulation of tonus, may account for the absence of decerebrate rigidity. At no point in the account of any of these experiments does he mention walking as one would expect if most of the righting reflexes were absent. In a note to Spatz,[28] Rademaker said, "dass sich Mittelhirnkatzen kriechend fortbewegten; also zum normalen Fortbewegungsakt waren sie offenbar nicht imstande." It is evident that the midbrain cats do not perform the complete act of progression as the midbrain rabbits have been seen to do. It is possible that the structures which are essential to this complex extend further rostrad in the cat and are injured by this plane of section, which in the rabbit leaves them functionally intact.

Rademaker came to the conclusion that the red nucleus is the chief center for the regulation of normal muscle tonus, the labyrinthine righting reflexes and the body righting reflexes acting on the body; that normal muscle tonus may still exist after complete removal of the cerebral hemispheres with the corpora striata, optic thalami and the oral portion of the red nucleus and after removal of the corpora quadrigemina; and that in the cat and the rabbit, a normal distribution of muscle tonus is no longer possible after removal of the red nuclei, after section of the decussation of Forel, even if the rest of the central nervous system is intact. He believed that there are at least seven systems which play a rôle in muscle tonus: (1) the red nucleus; (2) the center for decerebrate rigidity, which lies caudal to the pons; (3) the center for tonic labyrinthine reflexes, which is caudal to a plane just in front of the eighth nuclei and may coincide with the center for decerebrate rigidity; (4) the mechanism for tonic neck reflexes, the center for which lies in the rostral portion of the cervical spinal cord; (5) a spinal cord mechanism; (6) the proprioceptive stimuli, which may act on the center for decerebrate rigidity or on the spinal cord mechanism, and (7) the pyramidal tracts, as rigidity following section of the decussation of Forel is not so great with intact cerebral hemispheres as after their removal. The rôles of the corpus striatum, sub-

28. Spatz, H.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1927, vol. 10, pp. 318-417.

stantia nigra and cerebellum are not included in this scheme because he feels that their functions are unknown. Rademaker reviewed the cases with lesions of the red nucleus in man and reported that when there is unilateral or bilateral involvement of the red nuclei, there is an abnormal distribution of muscle tonus with hypertonia, ataxia and disturbances in posture. He concluded (p. 293), "Daraus folgt, dass die roten Kerne beim Menschen gleichartige Funktionen haben wie bei Katze und Kaninchen."

Laughton [15] experimented on rabbits, cats and dogs and came to the conclusion that the nervous mechanism which is involved in making possible the coordinated movements of progression is so situated that in the cat and the dog the caudal two thirds of the thalamus must be left if this mechanism is to function. In the rabbit, the cephalic two thirds of the pontile region is necessary, and the cerebellum is not essential. Medial longitudinal section through these portions of the brain stem eliminated the coordination of the movements of progression. He did not describe the complete act of walking in strictly midbrain rabbits, but he stated that in the thalamus rabbit a normal sitting posture was assumed and that in only a few instances did the animals show spontaneous efforts to run or hop. If they were stimulated by pinching the toes, tail or perineum, they hopped about the room and maintained their equilibrium perfectly. Thalamic cats could walk normally, with good coordination, but they were unable to avoid objects. If the section passed through the caudal third of the thalamus dorsally and through the cephalic end of the mammillary bodies ventrally, the rigidity was intense and at no time during the experiment did spontaneous movements occur. Faradic currents applied to the dorsal cutaneous nerves failed to elicit the alternate rhythmical movements in the fore legs throughout the experiments. However, after about thirty minutes, this stimulus brought forth alternate rhythmical movements in the hind legs. With this level of section, the cats were not shown to walk. Section of the brain stem rostral to the superior colliculi dorsally and just rostral to the roots of the oculomotor nerves ventrally resulted in a marked rigidity in all the limbs. In dogs, the complete act of walking was not observed, not even in dogs with thalamic preparations. However, stimulation elicited coordinated movements of progression as long as the plane of section was through or rostral to the superior colliculi dorsally and the point of exit of the oculomotor nerve ventrally; the rigidity was intense and there was a clonus in the hind limbs.

Schaltenbrand and Girndt [29] prepared thalamic cats in such a manner that the striatum was nearly completely removed. Some hours after

29. Schaltenbrand, G., and Girndt, O.: Arch. f. d. ges. Physiol. **209**:333, 1925.

the operation, the animals could walk and run without falling. The activity of these cats was characterized by periods of spontaneous movements with intervening periods of rest. During the period of activity, the breathing was accelerated and deepened, the eyes were opened, and there was a pronounced tonus of the extensors. The righting reflexes were present and well developed. During the period of rest, which lasted from ten to twenty minutes, the eyes were closed, the breathing was more quiet, the tonus of the extensors was decreased, and the righting reflexes were weak or could not be elicited. The acceleration and deepening of respiration were seen to precede the movements, and some animals showed an increase in the tonus of the muscles several seconds before the spontaneous movements began. When the animals were supported in a hammock with the limbs pendent, the tonus changes could be observed. During the periods of rest, the knee and elbow joints were slightly flexed. There were changes in the muscle tonus and breathing unaccompanied by movements of the limbs. In two experiments, the brain stem was sectioned between the superior and inferior colliculi during periods of movements. Decerebrate rigidity appeared, and the movements were no longer present. Girndt [30] said that the distribution of tonus in a thalamic cat is similar to that in the normal intact animal, but "aufgabe weiterer Forschung wird es sein, festzustellen, ob durch das Eingreifen der Pyramidenbahn und anderer corticospinaler Verbindungen die Reaktionsweise des intakten Tieres sich von der des Thalamustieres unterscheidet."

Pollock and Davis [31] ligated the basilar artery just posterior to the pituitary fossa, and the carotid arteries were then tied. The animal thus prepared attempted progressive movements. In the cage, it would crawl along the wall, spring forward and clasp the wire net of the cage. Sometimes it would crawl up the net until the head became extended, following which the fore legs would become rigidly extended and the animal would finally topple backward on the floor. Here these experimenters were dealing with a decerebrate animal, which exhibited the attitude of decerebrate rigidity in the extension in the fore legs; at times, it had normally distributed tone; sometimes there was decerebrate rigidity in flexion in the fore limbs; and sometimes there were spontaneous crawling, springing and some righting movements. Their work showed that decerebrate rigidity in extension or flexion and righting movements may occur whether the superior or the inferior parts of the cerebellum is functionless.

Mussen [32] was able to destroy the magnocellular portion of the red nucleus on both sides in the cat. The only symptom was a slight

30. Girndt, O.: Arch. f. d. ges. Physiol. **213**:427, 1926.
31. Pollock, L. J., and Davis, L.: Brain **50**:277, 1927.
32. Mussen, A. T.: Brain **50**:313, 1927.

unsteadiness of gait which lasted two days. During the remaining eighteen days, the gait was normal and the reflexes, sense of position and muscle tone were not affected. Then a lesion was placed in the anterior pole of the nucleus on the left side in another cat. On the first day, the animal was found in a sitting posture, the head body and tail curved to the right. On moving, it turned in a circle to the right, pivoting on its hind legs. There was a loss of sense of position in the left forepaw. Muscle tone and tendon reflexes were diminished. The "righting reflexes" were completely lost; the animal could be placed in any position, right side, left side or on its back, and it would remain as placed. From the fifth day on, the condition generally improved, the disturbance in the righting reflexes on the right side gradually disappeared and through the second and third weeks the only symptoms that remained were some unsteadiness in balancing and at times a slight turning of the head to the right. Mussen pointed out that there is a difference between symptoms produced by lesions in normal animals with otherwise intact nervous systems and those seen in thalamic and decerebrate animals. This probably explains the differences between his work and that of Magnus and his school.

The experiments of Bard [33] showed that if the caudal half of the diencephalon remained connected with the lower portion of the brain stem, a condition which he called sham rage developed. The manifestations of this arose spontaneously or could be elicited by stimulation.

They included struggling, attended by movements of the head and arching of the trunk with thrusting and pulling of the limbs; clawing movements of the forelegs with protrusion of the claws; waving and lashing of the tail; a snarling expression; a very rapid panting with mouth open and movements of the tongue to and fro. In addition to these activities, were signs denoting a vigorous sympathetic discharge: erection of the tail hairs; sweating from the toe pads; retraction of the nictitating membranes; exophthalmos (separation of lids); large increments in arterial pressure and heart rate.

During these observations, the cat was placed in a dorsal position on the animal board, secured by all four feet with the head slightly elevated. A cannula was inserted into a femoral artery for blood pressure recordings. Four of twelve cats in which only the hypothalamus remained (with only a small portion of the ventral and caudal parts of the thalamus intact) exhibited sham rage. Animals that were sectioned at a plane extending through the rostral borders of the superior colliculi dorsally and just behind the mammillary bodies ventrally exhibited rigidity throughout the experiment. Bard believed that there is a widespread sympathetic discharge accompanying sham rage, and that the diencephalic representation of the sympathetic nervous system consists of the mechanisms responsible for this sympathetic discharge. It will

33. Bard, P.: Am. J. Physiol. **84**:490, 1928.

be seen later that the somatic component in these fits of sham rage is exceedingly important and probably has its center in the mesencephalic tegmentum and the latter's continuation into the hypothalamus.

The typical decerebrate animal with the level of section through the mesencephalon does not possess any of the diencephalon. It is characterized by an exaggerated reflex standing, a condition known as decerebrate rigidity. It has been pointed out that movements of progression may be exhibited by an animal with this sort of preparation, but it does not perform the complete act of walking. Sherrington [34] stated that the stepping movements shown by the decapitate animal are amplified in the decerebrate animal into the performance of actual walking and running, which is "imperfect, it is true, especially in regard to equilibrium, the regulation of which is almost entirely wanting but nevertheless amounting to a certain measure of effective locomotion." As we shall later show, it would be important to know the exact level of section in an animal thus prepared. Magnus and de Kleijn [19] decerebrated cats at a level between the superior and inferior colliculi. In examining the effect of the position of the head on the distribution of tonus in muscles of the extremities, they found that well defined alternating running movements were superimposed on the tonicity of the limbs. These never occurred with minimal tonus of muscles of the extremities but only with medium to maximal tonus. Beritoff [35] believed that in the decerebrate animal there is present the capacity for locomotion, as it possesses the phasic stepping reflex and static tonus. He stated that if the decerebrate animal is brought into a standing position, with its feet on the floor, it not only will stand but will walk and move forward normally, but "nur ist die Fähigkeit zum Einhalten des Gleichgewichts beim Gehen sehr schwach, und einem Fallen vorzubeugen, muss das Präparat die ganze Zeit gehalten werden." Again it is seen that these animals do not possess equilibration and cannot maintain an upright position without support. We have no information in regard to the exact level of these sections. In the work of Bazett and Penfield [36] the levels of sections were accurately controlled and they stated (p. 222):

> Spontaneous movements in the form of progression as well as movements of the pseudoaffective and defence type have already been described, and it is clear that a distinction must be drawn between the progressive movements and pseudoaffective responses found after a section through the inferior colliculi to just in front of the pons and those seen when the section was 1 to 3 mm. more anterior, especially on the ventral aspect of the brain stem. In the former, the pseudoaffective responses usually developed only after two to four days, and

34. Sherrington, C. S.: J. Physiol. **40**:28, 1910.
35. Beritoff, J. S.: Arch. f. d. ges. Physiol. **199**:248, 1922.
36. Bazett, H. C., and Penfield, W. G.: Brain **45**:185, 1922.

though they gradually became more active (e. g., cat II) they were never violent. In the latter (as in cat XIX) these movements were seen soon after operation and were much more violent.

The latter movements recurred in paroxysms every fifteen to forty-five minutes. Immediately after the paroxysm there was a quiescent period during which the animal lay passive in a condition of simple decerebrate rigidity.

From the evidence as it has been reviewed, it appears that in the rabbit the mechanism for walking is fairly complete in the brain stem below the plane dividing the mesencephalon from the diencephalon. In the cat, on the other hand, a plane of section at this level leads to decerebrate rigidity. This was shown in all experiments, except those of Rademaker,[27] which are not convincing. Either the effect of shock is greater in the cat than in the rabbit or the mechanism in the cat projects up into the diencephalon and is injured by this plane of section. In the monkey, the experiments have been so inconclusive that it is impossible to say what is the essential mechanism of reflex walking.

METHOD

Our experiments have been performed on cats and rabbits. The animal was anesthetized with ether and the carotid arteries were ligated in the neck; as a general rule, a double tie was made above and below the superior thyroid artery. A tracheal cannula was inserted, and the wound was then closed. The calvarium was exposed by a transverse incision and opened with a drill and rongeur forceps over the occipital poles of both cerebral hemispheres. The opening was extended so that a large portion of the dorsal and lateral surfaces of the cerebral hemispheres was exposed. The dura was opened by a transverse incision, after the superior sagittal sinus had been doubly ligated and cut between the ligatures. The dura was cut in a rostral and caudal direction over each hemisphere at right angles to the transverse incision. Bone bleeding was controlled by bone wax and dural bleeding by small pieces of muscle. The occipital pole of each hemisphere was raised, and a sharp-edged spoon was used to remove each hemisphere. Then a sharp knife was employed to make the transection at the desired level. All of the brain rostral to this section was removed and the blood sponged away with cotton until hemorrhage ceased. The cranium was not packed with cotton, nor were the vertebral arteries compressed at any time. Compression of these arteries, following ligation of the carotid, deprives the medulla oblongata of blood, causes interference with respiration and is likely to produce shock, which would delay and interfere with the appearance of the reactions in the animal. During the time when the cerebral hemispheres were being removed and the brain stem sectioned, the animal board was raised so that the head would be lower than the rest of the body, thus preventing an oozing of blood into the subpontile region. Since the consciousness of pain was no longer present in these animals, the ether was discontinued following section of the brain stem. A subcutaneous injection of 50 cc. of physiologic solution of sodium chloride was then made.

The behavior of the animal was constantly observed throughout the experiment, which lasted from five to eight hours. The rectal temperatures were

recorded from time to time and the body temperature was maintained as close to 37 C. as possible. Our main interest throughout was in the righting reflexes and in walking, together with the distribution of tonus in the musculature.

At the end of the experiment, the animal was killed. The remaining brain stem was removed and placed in a solution of neutral formaldehyde, U. S. P. (1:10). The brain stems were sectioned and stained by Weil's[37] method for myelin sheath stains and with cresyl violet for cellular differentiation.

OBSERVATIONS

If the plane of section passed from the rostral border of the superior colliculus to the region of the optic chiasm (fig. 1), the animal on cessation of the anesthetic first showed decerebrate rigidity. In cat 12 was a typical example of this type of preparation. Fifteen minutes after the operation was completed there was a definite tendency on the part of the animal to right itself, but at this time the reaction was

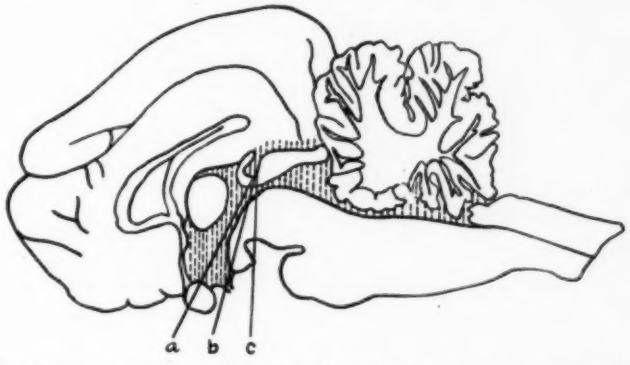

Fig. 1.—Median sagittal section of the cat's brain showing the location of the planes of transection: (*a*) through the optic chiasm; (*b*) through the rostral portion of the mammillary bodies, and (*c*) just caudal to the mammillary bodies.

incomplete. Thirty minutes after the cessation of the anesthetic, the animal could right itself and walk with its hind limbs in normal posture, but in the fore limbs there was a tendency for it to walk on its wrists. Walking was carried out with normal coordination, and the animal maintained its upright posture exceedingly well. Two hours later, this tendency to walk on its wrists had almost completely disappeared. From this time on, there were periods of spontaneous righting reflexes and walking with varying intervals of rest between. During the periods of activity, the respiration rate was accelerated and the respirations deepened, and this condition continued for varying intervals of time following the activity. During the periods of walking, the animal progressed aimlessly about the room, generally in a fairly straight line,

37. Weil, A.: Rapid Method for Staining Myelin Sheaths, Arch. Neurol. & Psychiat. **20**:392 (Aug.) 1928.

but at times in circles. When it met an obstacle, the wall, for example, it would continue in an attempt to move forward until reflexly it would turn, sometimes follow the wall for a distance and then wander away from it. In some instances, when an obstacle was met, the progress of the animal would be completely arrested. The cat could climb on muslin stretched over an upright wire frame and one time climbed to the top, a distance of 3½ feet (107 cm.). There was a disturbance of the heat-regulating mechanism, and artificial heat in the form of hot water bottles was applied to the animal during the periods of rest. It was necessary to put the cat under a canvas cover to apply the heat, and it would wriggle out from beneath the cover with ease. At the time the motion pictures were taken, the hyperextension of all limbs in the periods of rest could easily be seen with the animal on its side or on its back. As long as the animal was in activity or was in a resting crouching position, there was no marked predominance of extensor tonus. When such an animal was palpated while in the crouching resting position, one gained the impression that, in spite of the fact that there was a fairly equal distribution of tonus between flexors and extensors, there was an exaggeration in the tonus in both compared with the tonus of a normal animal at rest. The observations on cat 12 were continued for seven hours, and the condition of the cat was excellent when it was killed. In all, we found the righting and walking pattern present in fourteen cats sectioned at this level. The percentage of successful experiments was about 50. In the cats in which righting and walking were absent, the absence may be explained as due to the presence of hemorrhage and shock, which are exceedingly difficult to avoid in experiments of this kind.

In speaking of the nuclear masses in this region of the mesencephalic tegmentum and its continuation into the hypothalamus, we shall follow the terminology used by Winkler and Potter [7] in their atlas of the cat's brain, because it is accessible and has been followed by others working in this same field. There are differences of opinion in regard to the terminology to be applied to these nuclear masses, and our use of the Winkler-Potter terminology need not be interpreted to mean that we subscribe to its correctness. We merely endeavor to avoid confusing those who are not acquainted with the intricate details of the terminology applied to this portion of the brain stem.

Whatever nervous mechanism is responsible for the righting and walking pattern in the cat resides caudal to a plane extending from the rostral border of the superior colliculus to the region of the optic chiasm. Following section in a plane of this kind, there is present an extremely caudal portion of the optic thalamus, there may be some portion of the pyriform lobe left attached, and in between the optic tract and the

cerebral peduncle there is a nuclear structure which Winkler called globus pallidus. The latter structure has been called the entopeduncular nucleus and also the olfactory striatum or peduncular nucleus. In addition to these structures, there is the continuation of the mesencephalic tegmentum up into the subthalamic portion of the hypothalamus. In endeavoring to remove the group of cells that Winkler and Potter called globus pallidus, we made two series of experiments; (1) with the plane of section extending from the rostral border of the superior colliculi to the mammillary bodies, and (2) with the plane of section extending dorsally through the posterior third of the thalamus several millimeters in front of the habenular commissure to the mammillary bodies ventrally.

In cats 17 and 19, the righting and walking patterns were obtained with the plane of section extending through the rostral portion of the superior colliculi to the rostral border of the mammillary bodies (fig. 1). Cat 17, thus prepared, exhibited spontaneous righting and walking, but this animal did not show the periodic activity that cat 12 did. The righting and the walking pattern were complete in this preparation, and there was an involvement of the fore limbs so that the cat walked on its wrists. In this preparation, the extreme caudal tip of the optic thalamus was present just medial to the medial geniculate body. The globus pallidus and cerebral cortex were completely removed. The corpus subthalamicum (peripeduncular nucleus of Jacobsohn) and the nucleus proprius pedunculi cerebri (subthalamic nucleus of Luys) were sectioned through their upper third. The red nucleus was completely intact. Cat 19 showed essentially the same reactions that were seen in cat 17. Both exhibited the so-called "normal tonus" while in the resting crouching position and while walking. They were both less active and lacked the periodicity of movement seen in some of the cats with the plane of section at the same level dorsally and through the optic chiasm ventrally. However, the walking and righting complex in each was complete.

When the section passed through a plane 4 or 5 mm. in front of the habenular commissure to the rostral border of the mammillary bodies ventrally, the animals walked and righted themselves spontaneously, but here the movements were not so periodic. Cat 31 was much less active than cat 34 in spite of the fact that the level of section was essentially the same. The globus pallidus was completely gone in both, the red nucleus was intact, and most of the corpus subthalamicum (peripeduncular nucleus of Jacobsohn) and of the nucleus proprius pedunculi cerebri (subthalamic nucleus of Luys) was present. In cat 31, the cerebral cortex was completely removed, and in cat 34 a portion of the pyriform lobes was left on each side.

In cat 32, the section passed through a plane extending from 7 mm. in front of the habenular commissure to the mammillary bodies ventrally. The cerebral cortex was completely removed in this cat, but a small portion of the globus pallidus was left on one side. This animal righted itself, walked well and showed marked periodicity of the spontaneous movements. This animal was not fed the day before the operation.

The thalamic cats behaved much the same as the hypothalmic ones in respect to righting and walking. The protocol for cat 40 is typical.

When the section passed from the rostral border of the superior colliculi to just behind the mammillary bodies in front of the exit of the oculomotor nerves (fig. 1), the cat generally showed the typical con-

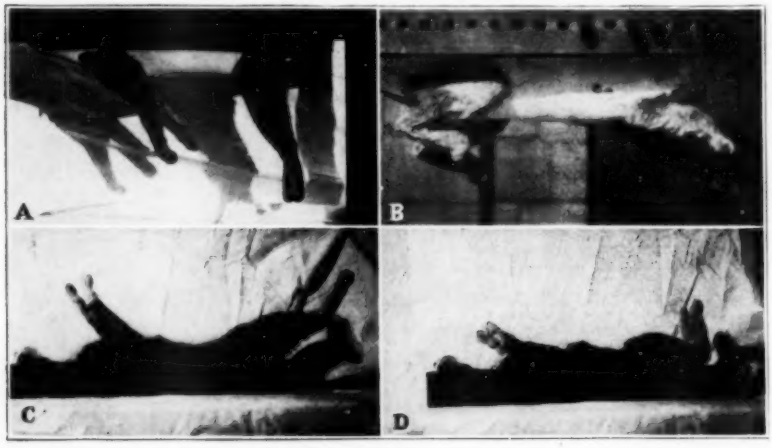

Fig. 2.—*A*, a posterior thalamic cat (no. 33) showing hyperextension in the limbs when it is placed in the hammock. *B*, a normal cat in the hammock. *C*, a shortening reaction in cat 43, which was sectioned from the rostral border of the superior colliculi to just behind the mammillary bodies. *D*, a lengthening reaction in cat 43.

dition of decerebrate rigidity. Some of the cats prepared in this way, for example cat 43, showed spontaneous poorly coordinated running movements, but the righting reflexes were imperfect, and the cat did not walk. These animals were less stiff than those in which the section was made through the rostral border of the pons; but, whether the animals were placed on their backs or suspended by strings passing through the muscles and ligaments on the dorsal side of the vertebral column, they showed decerebrate rigidity of all four limbs, plasticity and the lengthening and shortening reactions (figs. 2C and D). If placed on their sides, they remained quiet for long periods of time with the limbs held in an extended posture. As a general rule, there was no indication

of the righting reflexes being present, but one sees from the protocol that they may be present, imperfectly developed. The mechanism responsible for the complete act of walking was either partially removed in these sections or so injured that it could not function.

From the evidence here presented, it is plain to be seen that whatever nervous mechanism is involved in the complete act of walking in the cat is situated caudal to a plane of section of the brain stem extending from the rostral border of the superior colliculi dorsally to the rostral border of the mammillary bodies ventrally (fig. 1). It is impossible to say definitely which centers are involved, but it is tempting to

TABLE 1.—*Showing Some of the Parts of the Brain Which Remained Intact in Walking Cats*

Animal	Walking	Pallidum	Red Nucleus	Corpus Subthalamicum	Nucleus Proprius Pedunculi
11	++	Present, R & L	Intact	Intact	Intact
12	++	Present, R & L	Intact	Intact	Intact
13	0	Absent, R & L	Lower half intact	Absent	Absent
14	0	Present, R & L	Intact	Intact	Intact
15	+	Present, R & L	Intact	Intact	Intact
16	+	Present, R & L	Intact	Intact	Intact
17	+	Absent, R & L	Intact	Lower two thirds present	Lower two thirds present
19	+	Absent, R & L	Intact	Lower two thirds present	Lower two thirds present
20	++	Present, R & L	Intact	Intact	Intact
22	++	Present, R & L	Intact	Intact	Intact
25	+	Trace on one side	Intact	Intact	Intact
26	+	Present, R & L	Intact	Intact	Intact
27	+	Present on one side	Intact	Intact	Intact
30	++	Present, R & L	Intact	Intact	Intact
31	++	Absent, R & L	Intact	Intact or perhaps just upper end gone	Through upper end
32	++	Present on one side	Intact	Intact	Intact
34	++	Absent, R & L	Intact	Lower two thirds present	Lower two thirds present
43	0	Absent, R & L	Lower half intact	Absent	Absent

associate the mechanism with the mesencephalic tegmentum and its continuation into the hypothalamus including the red nucleus and other centers present. As the accompanying table shows, in all cats that walked, the red nucleus was completely intact together with at least two thirds of the nucleus proprius pedunculi cerebri and the corpus subthalamicum. The interstitial nucleus of Cajal was left, as was the nucleus of Darkschewitsch.

We have made sections through the brain stems of rabbits and have confirmed the work of Rademaker and Magnus for the thalamic animal, as is illustrated in the protocol of rabbit 38. If the plane of section extended from the rostral border of the superior colliculi to the caudal

portion of the mammillary bodies, the animal was similar to those given the thalamic preparation. In one case in which the plane of section extended from the rostral border of the superior colliculus to a point just rostral to the exit of the third nerve, the animal showed complete righting reactions with normal distribution of tonus in the crouching position, but failed to hop when stimulated. It was necessary to stimulate even the thalamic rabbit in order to cause it to hop.

The use of the word spontaneous may be misleading, for to some it may suggest movements of a voluntary nature. However, spontaneous, as here used, means that there was no obvious external stimulation. The possibility of stimulation of an internal nature is of course not eliminated in any of these experiments. Visceral stimulation may be the cause of these so-called spontaneous movements. Dresel [6] found that his dog became extremely restless when hungry or when the bladder was distended. In the opossum, Rogers [8] observed that when the cerebral hemispheres were removed entire, the animal showed periodic activity with intervening periods of sleep. This restlessness could be neither initiated nor inhibited by olfactory, visual, auditory or cutaneous stimulation, but in certain cases it was definitely inhibited by feeding. In our experiments, the periodic activity that was observed may well have been due to some visceral stimulation. While the thalamic and hypothalamic cats showed this condition more commonly than the cats sectioned through the superior colliculus and through the mammillary body, it is to be noted that the cat (43) sectioned from a point at the rostral border of the superior colliculus to just rostrad to the point of exit of the oculomotor nerves showed periodic spontaneous running movements. In cat 32, which was brought into the laboratory on November 27 and not fed all that day and operated on the following day, these spontaneous movements were markedly developed. From our data it would be impossible for us to associate the spontaneous movements with different levels of section, because it is impossible to have the different animals in the same physiologic condition at the time of the experiment, but the movements may occur in the complete absence of the neostriatum and paleostriatum. It is an interesting fact that the movements were entirely absent in the rabbit. In the cat, they were nearly always associated with accelerated and deepened respirations. In the periods of rest, the eyes were sometimes closed and sometimes opened.

As has been seen, Rademaker believed that the red nucleus is a center governing the normal distribution of tonus. He found that in the thalamic and so-called midbrain cats, the distribution of tonus was normal. It is a difficult task to define normal tonus. Magnus [25] said that, if the midbrain is left intact (p. 4), "erstens schwindet die Enthirnungsstarre, die einseitige Bevorzugung der Streckmuskeln fällt

fort, und statt dessen tritt eine normale Tonusverteilung zwischen den Streck- und Beugemuskeln auf, gerade so, wie das beim normalen intakten Tiere der Fall ist," and that not only the extensors but the flexors have their normal tonus. But how is one to judge what the normal tonus would be in the extensors of a cat in the waking condition? One standard that might be taken would consist of the tonus found in a normal human being at complete rest in the waking state, as when tonus is tested clinically. But here one cannot exclude the possibility of a voluntary inhibition of tonus. Is one to accept the absence of a predominance of extensor tonus or of flexor tonus with a fairly equal distribution between the two when at rest as normal tonus in the cat? It would be possible to have fairly well executed movements and still have an exaggeration of tonus in both flexors and extensors. Furthermore, in certain postures, the tonus might be similar to normal and in others there might be a predominance of either extensor or flexor tonus. It would seem reasonable to assume that, if in any posture, the distribution of tonus differed from that seen in a normal, intact animal, one should not speak of the animal as showing normal distribution of tonus, but should qualify one's remarks by stating in what postures it was found. The absence of decerebrate rigidity per se does not permit one to speak of tonus as being normal.

The thalamic cat, the hypothalamic cat (with the plane of section extending as far caudally as the rostral border of the superior colliculus and through the rostral portion of the mammillary body), the thalamic rabbit and the midbrain rabbit (with the plane of section extending from as far caudally as the rostral border of the superior colliculus dorsally to a point just rostral to the exit of the third nerve ventrally) show what might be called normal tonus as long as they are in the resting crouching position or standing or walking. There is an absence of a predominance of either flexor or extensor tonus beyond what would be expected in a normal cat under similar conditions. However, when the animal is at rest in a crouching position, one gains the impression, on palpation, that there is an exaggeration of both flexor and extensor tonus beyond what would be present in a normal animal. When one of these animals is supported in a hammock, as shown in figure 2A, all the limbs show an exaggeration of extensor tonus, more marked in the hind limbs in some cases than in the fore limbs. Protocols of thalamic rabbit 38, thalamic cat 40, and posterior-thalamic cat 34 illustrate this point. A normal cat at rest in this hammock tends to show flexion in both fore and hind limbs, as is illustrated in figure 2B. Motion pictures of the hypothalamic cats show distinctly that if the animal is on its side or its back and is palpated, there is a definite resistance to passive flexion. In the motion pictures of cat 22, the position of hyperextension in both fore and hind limbs was seen when

the animal was supported by holding it up by the skin in the dorsal midline. A thalamic cat which had righted and walked was supported by three strings passing through the vertebral ligaments, and the head was supported by a rigid transverse bar. With the cat in this position, we found resistance to passive flexion in all four limbs even when the head was maintained in normal position with respect to the rest of the body. It is evident from these observations that in our prepared animals which righted and walked, both cat and rabbit, in certain postures there was a nearly "normal" distribution of tonus, and in other postures, a marked hypertonicity of the extensors. One interpretation that might be given is that the nervous mechanism responsible for the regulation of tonus is in certain postures able to inhibit the hypertonus of the extensors which is present in the other postures. The conclusion to be drawn is that it is impossible to say that in the animals such as we have described there is a normal distribution of tonus in all postures.

A weakness of the muscles of the wrist joints in the fore limbs of the cats that walked led to a tendency to walk on the wrists. About two thirds of our series showed this, and in one half of these it persisted throughout the experiment. This condition was never observed at the ankle joints in the hind limbs. It is seen in a number of different types of experiments, i. e., in decorticated cats (Dusser de Barenne [3]), cats in which the area frontalis has been removed (King [14]), thalamic (Magnus [25]) and hypothalamic cats and cats in which the fore limbs had been deprived of the afferent nerves. Whether it is to be attributed to a loss of the sense of position, or to some disturbance of the motor pathway causing a predominance in the tonus of the flexors of the wrists, it must be considered as an abnormality in distribution of tonus. The tendency to walk on the wrists was not seen in our thalamic and midbrain rabbits.

As already seen, if the plane of section passes from the rostral border of the superior colliculus to just behind the mammillary bodies, the cat shows decerebrate rigidity and does not walk (cat 43). To be sure, these animals are less stiff than when the section passes through the rostral border of the pons; nevertheless they show definite hypertonicity of the extensors. Figure 2C and D shows the lengthening and shortening reactions as seen in a cat in which the plane of section passes from the rostral border of the superior colliculus to the caudal border of the mammillary body. A large series of these was made, and decerebrate rigidity was always present with this plane of section (Ranson and Hinsey [38]).

38. Ranson, S. W., and Hinsey, J. C., to be published.

As our experiments were not primarily designed to ascertain a heat-regulating center, no conclusions in regard to this point can be drawn from them. If the section passes from the rostral border of the superior colliculi to just back of the mammillary bodies, the rectal temperature may be maintained at a normal level at room temperature for some time (protocol of cat 43). However, when such an animal is exposed to a lower temperature, it is unable to maintain its normal level. In other cats sectioned through a similar plane, the normal level was not maintained, even at room temperature. When the section passed from the rostral border of the superior colliculus through the rostral portion of the mammillary body (protocol of cat 17), the temperature was not maintained at a normal level. The same is true of most hypothalamic preparations with the level of section from the rostral border of the superior colliculus to the region of the optic chiasm (protocol of cat 12). In cat 20, the temperature was maintained fairly well. In cat 34, with the plane of section extending 4 mm. in front of the habenular commissure dorsally and through the rostral part of the mammillary bodies ventrally, the temperature tended to fall. In cat 32, with a similar level of section, the temperature was maintained excellently. The protocol shows that the temperature was maintained in spite of exposure to a room temperature of 17 C. This animal was an extremely active one. The protocol of cat 40, with a thalamic preparation, shows that the normal body temperature was maintained fairly well. The results are so variable that it is impossible to draw any conclusions, but the general trend of the observations seems to indicate that the animal with a hypothalamic preparation, i. e., with the plane of section extending from the rostral border of the superior colliculus to the region of the optic chiasm, is unable to maintain its normal body temperature (protocol of cat 12).

These experiments were not planned in order that we might observe the reactions of sham rage and the sympathetic outflow that Bard [33] described. Our animals were not tied down in the dorsal position on the animal board and no blood pressure tracings were made. In one thalamic cat (Jan. 3, 1929), when it was tied in the dorsal position on the animal board, there were spontaneous convulsive attempts to free itself accompanied by ruffling of the hair and acceleration and deepening of respiration but with no sham rage in the face or perspiration on the paws. Observations on perspiration should be made in an atmosphere with a high relative humidity, and for that reason, an absence in our experiments cannot be taken to mean that it does not occur. In cat 32, with the plane of section extending from 7 mm. rostral to the habenular commissure to the rostral portion of the mammillary bodies, there was an expression of rage in the face with an erection of the hair on the tail during activity. The protrusion of the claws in the

fore limbs occurred regularly as a part of the righting reflex. Lashing of the tail was seen in a great many of the walking cats. The somatic motor disturbances described in Bard's work were undoubtedly activities which, if the cats had been free, would have resulted in righting and walking. However, when hypothalamic sections were made extending from the rostral border of the superior colliculus to the region of the optic chiasm ventrally, the motion pictures do not show any particular evidence of sham rage in the faces of such animals when they were free to move about.

COMMENT

The difficulty in securing a definition or a standard of what is meant by normal tonus has been discussed. Our observations have shown that, when cats with thalamic and hypothalamic preparations are in the resting crouching position or standing or walking, there are no marked distortions of tonic distribution and the movements closely resemble normal movements. However, in some of them there was a temporary, and in others a permanent (as long as the experiment continued) involvement of the muscles of the fore limbs that control the normal posture of the wrists. This condition reminded us of the involvement in the fore limbs of the cats in other kinds of experiments already cited and should be considered as an abnormality in the distribution of tonus. When our animals were supported in the air by grasping of the skin in the middorsal line of the neck and sacral regions, in a hammock with limbs pendent or by strings drawn through the ligaments of the vertebral column, they showed marked hypertonicity of the extensor muscles in the fore and hind limbs (more marked in the hind limbs). In some of the preparations, this rigidity was broken through by progressive movements. When the limbs of these animals were palpated while in the crouching position, there was an exaggeration in the tonus of both the flexors and the extensors, which exceeded that of a normal human being in a state of complete relaxation.

The question resolves itself into whether or not a hypothalamic cat has a nervous mechanism competent to bring about a normal distribution of tonus. It would seem that when resting in the crouching position, or standing or walking engages the animal, the mechanism meets the situation and brings about a distribution that is similar to the normal. Rademaker himself admitted that the rigidity following removal of the influence of the red nucleus with an otherwise intact central nervous system is not so great as when the animal is decerebrated. From this it would seem that there is some inhibitory influence at a higher level than the one which he holds mainly responsible for normal distribution of tonus. The work that was described earlier would lead one to believe that there is an inhibitory influence in the cerebral cortex of

the cat. Whether this influence is in the motor area (Laughton, Bernis and Spiegel, and McKibben), in the area frontalis (Weed, Warner and Olmsted, Olmsted and Logan, King, Langworthy, Bernis and Spiegel), in the temporal lobe (Bernis and Spiegel) or in all these areas is a matter on which there is difference of opinion. All these experiments show, however, that there is an inhibitory influence somewhere in the cerebral cortex. The influence of the corpus striatum has been subjected to a large number of experiments which at the present time are difficult to evaluate (Spatz [28]). It is difficult to conceive of there being no disturbance of tonus distribution with the cerebral influence and the corpus striatum completely removed. This change in the distribution of tonus may not be evident in all postures and it may take special manipulation to show it, but there is no doubt that both the thalamic and the hypothalamic cats and the thalamic and midbrain rabbits show disturbances of tonus distribution in certain postures. We feel that the term "normal tonus" should be more strictly qualified in its application to these animals.

The observations made on the cat and the rabbit differ in regard to the conditions seen when the plane of section extends from the rostral border of the superior colliculus to just behind the mammillary bodies. Contrary to the contentions of Rademaker, but in agreement with those of other observers, we found that this plane of section was followed by decerebrate rigidity in the cat. In the rabbit, our observations were similar, in many respects, to those of Magnus and of Rademaker. This difference between the physiologic observations in the cat and those in the rabbit following section at this level may be subjected to at least three interpretations: If the influence of shock were greater in the cat than in the rabbit, a higher section might be required in the former than in the latter. It may be that in the cat the influence of a higher center has been brought into play which is removed by this plane of section. And then there is a possibility that essentially the same mechanism is involved but that its anatomic position with respect to the rest of the brain stem has been so changed in the cat that this level of section passes through it or injures it so as to prevent its functioning. The last explanation seems a plausible one, for Winkler's atlases [39] show that one of the structures in the mesencephalic tegmentum projects to a higher level in the cat than in the rabbit. However, the fact cannot be overlooked that the two animals vary in behavior even to the mode of progression, and it is perfectly conceivable that the combination of nerve centers employed in one animal may be different from that involved in the other.

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39. Winkler and Potter (footnote 7); An Anatomical Guide to Experimental Researches on the Rabbit's Brain, Amsterdam, W. Versluys, 1911.

Our experiments have shown that whatever nervous mechanism is responsible for the complete act of walking in the cat, it resides caudal to a plane of section extending from the rostral border of the superior colliculi to the rostral portion of the mammillary bodies. In this remaining brain stem are the nerve pathways for the three essential components of the complete act of walking: (1) rhythmicity; (2) static or postural tonus, and (3) equilibration. The synthesis of these components into reflex walking is complete in the absence of the cerebral cortex and the neostriatum and paleostriatum.

The experiments of Freusberg,[40] Philippson,[41] Sherrington,[34] Brown,[42] Beritoff,[35] Miller [43] and Laughton [15] on spinal animals have shown that the lumbar and sacral regions of the spinal cord give rise to rhythmic stepping movements, which may be elicited following stimulation or may occur spontaneously when the animal is supported in the upright position with the limbs pendent. Graham Brown [42] showed that the rhythmic stepping movements occurred in the hind limbs of the cat following deafferentation of both limbs, showing, contrary to the original view of Sherrington, that the movements occur in the absence of proprioceptive impulses from the muscles themselves. Brown believed that the rhythmic sequence in the act of progression is determined by phasic changes innate in the local spinal cord centers, and these changes are not essentially caused by peripheral stimuli. He thought that the proprioceptive impulses play a regulating and not an intrinsic part in the act. Sherrington [34] showed that, in the decapitate cat, stimulation in the various regions of the body brought forth reflex stepping in the hind limbs, but that the reflex responses were difficult to elicit in the fore limbs. The reflexes in the fore limbs were more depressed following decapitation than following decerebration, so that with the former a strong stimulus to the fore limb, though it evoked a reflex movement in the ipsolateral hind limb, might have called forth little or none in the fore limb. Beritoff [35] found that if a transection of the spinal cord was made in the first cervical segment, phasic movements of the fore limbs were never seen, owing, he thought, to a depression of the cervical cord. In the decapitate cat, Miller [43] applied nociceptive stimuli to the pads of the forepaws and observed reflex stepping in the hind limbs but not in the fore limbs. Laughton [15] studied the coordinated movements of progression in the rabbit with respect to coordination of the fore and hind limbs. He observed that

40. Freusberg, A.: Arch. f. d. ges. Physiol. **9**:358, 1874.

41. Philippson, M.: L'autonomie et la centralisation dans le système nerveux des animaux. Traveux du Lab. de Physiol., Institute Solvay, Bruxelles, published by P. Hegner, 1905, vol. 7, pt. 2, pp. 1-208. (Not seen.)

42. Brown, T. G.: Proc. Roy. Soc., London (ser. B) **84**:308, 1912.

43. Miller, F. R.: Tr. Roy. Soc. Canada **17**: sec. 5, 1923.

removal of the cephalic portion of the pontile region resulted in the elimination of the movements of the fore legs, but did not affect the synchronous bilateral movements of the hind legs. In the cat, he found that removal of the middle third and the cephalic portion of the caudal third of the thalamus resulted in the elimination of the coordinated movements in the fore legs while the hind legs were unaffected. He stated that coordinated movements in sequence in the hind legs and fore legs have never been described as occurring in decapitate animals. Whether or not this absence of coordination between the rhythmic movements of the fore and hind limbs is due to the removal of a physiologic mechanism situated at a higher level in the brain stem or to the presence of shock in the rostral portion of the spinal cord is a question which cannot be settled on the basis of the physiologic evidence at hand. *A priori* it would seem that the reflex pathways for such a coordination should be present in the spinal cord itself, for if the mechanism for the hind limbs is present in the lumbosacral cord, it would seem that a similar mechanism for the fore limbs should exist in the cervical cord.

Magnus and de Kleijn [19] were able to elicit alternating running movements in decerebrate cats by changes in the posture of the head and neck, but whether the movements in the fore limbs were coordinated with those in the hind limbs we do not know. Sherrington [34] stated that the stepping movements seen in the decapitate animals are amplified in the decerebrate ones (level not stated) into the performance of actual walking and running with almost complete lack of equilibration but with a certain measure of effective locomotion. Beritoff [35] also stated that if a decerebrate animal (level not given) is brought into a standing position and supported, it not only can stand but can move forward normally. Here again, coordination between fore and hind limbs is implied. Davis and Pollock [44] observed that after destruction of the labyrinths, phasic reflexes could be elicited more easily than following ordinary decerebrations. Running movements were pronounced and frequently were so active that it was difficult to balance the animal so that the position of standing could be imposed. They were of the opinion that the labyrinths reenforce the extensor muscles of the neck and body, with a consequent rigidity in the fore legs so great as to prevent the appearance of certain phasic reflexes.

In the protocol of cat 43 with the plane of section extending from the rostral border of the superior colliculus to just rostral to the point of exit of the oculomotor nerves, it may be seen that the animal showed spontaneous movements of both the fore and the hind limbs. In the fore limbs, the movements were alternating, and the hind limbs were

44. Davis, L., and Pollock, L. J.: Studies in Decerebration; Labyrinth, Arch. Neurol. & Psychiat. **16**:555 (Nov.) 1926.

brought forward in what resembled a jumping movement, coordination being poorly developed. We know that if the plane of section passes from the rostral border of the superior colliculi through the rostral portion of the mammillary bodies ventrally there is perfect coordination between the movements of the fore, and those of the hind limbs in the complete act of walking. However, we do not feel that the evidence as yet necessitates a prespinal mechanism, and not until another approach to the problem is made will the evidence be convincing on this point. According to accounts in the literature, the decapitate animals show depression of the upper cervical spinal cord, and in this region may be found a mechanism which, if functional in an undepressed state, is responsible for the rhythmic alternating movements of the fore limbs and, in addition, coordination between the fore and the hind limbs.

The second component of the complete act of walking is static or postural tonus. Sherrington [34] stated that the decapitate cat cannot stand. If placed in an erect posture, it sinks to the table, and the muscles of the limbs, neck and tail are unable to antagonize gravity. However, if a dog's spinal cord is transected at the level of the tenth thoracic segment (Philippson [45]) and the animal is observed some months or weeks later, the hind limbs are found capable of maintaining the extended posture and supporting the weight of the posterior part of the body, even for minutes at a time. The animal is able to stand on three legs, using only one hind limb. Spinal standing was found to be subject to lapses. Beritoff [35] reported that in cats, when the spinal cord is transected beneath the medulla oblongata, there is extensor tonus in all four legs which is more pronounced in the hind limbs. It appears thirty minutes after the operation and may be present as long as from five to twelve hours. The hind limbs are said to be able to support the body weight for from ten to twenty seconds. According to this evidence, there is a part of static tonus that is due to some mechanism situated in the spinal cord. However, an animal sectioned between the superior and inferior colliculi shows this type of tonus so much exaggerated that it may be said that reflex standing engages the animal and it is said to exhibit decerebrate rigidity. This exaggeration of static or postural tonus is due to the activity of some mechanism in the medulla oblongata which is held under inhibition by centers situated in the mesencephalic tegmentum and its continuation into the hypothalamus. It has been seen in the protocol of cat 43 that the tonus was exaggerated. It becomes more so as the plane of section is more caudal in the mesencephalon (Ranson and Hinsey [38]).

The third component of the complete act of walking is equilibration and the ability to maintain an upright posture. Here we have to deal with the righting reflexes, which Magnus and his co-workers analyzed

45. Philippson, M.: International Congress of Physiologists, Heidelberg, 1907, p. 130, quoted from Sherrington (footnote 34, p. 103).

into five types in the cat: (1) optic, (2) labyrinthine, (3) body reflexes acting on the body, (4) body and head reflexes acting on the head and (5) neck righting reflexes. The optical righting reflexes are not present in the thalamic animal. The remaining four are present in a thalamic animal and are essential to this third component of the complete act of walking.

The labyrinthine righting reflexes are evoked from the otolithic apparatus, and they serve to keep the head in the normal position. Their center is said to be in the red nucleus. The body reflexes acting on the head have their center in the midbrain at the level of the red nucleus but are said not to pass over the red nucleus (Rademaker,[27] p. 292). The neck righting reflexes have their center caudal to the rostral border of the pons. The body righting reflexes acting on the body have their center in the red nucleus and are said to be absent following decerebellation (Rademaker,[27] p. 220). From this analysis of the righting reflexes which Magnus and his co-workers have made, it is evident that the mesencephalic tegmentum, including the red nucleus and other tegmental structures about it, is essential to the righting component and should be necessary for the complete act of walking. The labyrinthine righting reflexes, the body reflexes acting on the head, and the neck righting reflexes are all present following cerebellar extirpation, while the body righting reflexes acting on the body are said to be absent. However, Magnus, in his Croonian lecture,[46] stated, "Our evidence of the postural activity of the cerebellum is purely negative."

The decerebrate animal with the level of section through the mesencephalon between the superior and inferior colliculi has no righting function. The animal is able to stand when placed in position, but if it is pushed over or falls over, it lies there and is unable to right itself and return to a normal standing position. In our experience, the cat with the level of section from the rostral border of the superior colliculus dorsally to just behind the mammillary bodies ventrally is unable to show the complete righting pattern and exhibits decerebrate rigidity. However, an animal sectioned a little higher up on the ventral surface, through the rostral portion of the mammillary bodies, shows complete righting and walking, during which the distribution of tonus is nearly normal. As we have stated before, Rademaker [27] believed that the red nucleus is the center for normal tonus distribution, for labyrinthine righting reflexes and for body righting reflexes acting on the body. However, it would be impossible for us to say that the red nucleus is the only structure in this upper portion of the mesencephalic tegmentum that is essential for the complete act of walking.

It will be impossible to review the physiologic evidence in relation to the question of the control of posture, but the question has recently been

46. Magnus, R.: Proc. Roy. Soc., London (ser. B) **98**:339, 1925.

reviewed by Spatz [28] and by Langworthy.[47] In our work, we have been interested in what nervous mechanisms we could remove and still have the complete act of walking present. Figure 3 is a diagram showing some of the prespinal mechanisms which may be involved in the control of posture and of walking. We do not mean to imply that all these mechanisms exert their influence directly on the final common path; in fact, knowledge in regard to how these prespinal mechanisms act on the neurons of the final common path is negligible. The number of the mechanisms recognized as having such action is by no means complete, and some of those included in the diagram may have little or no influence. The figure shows in a diagrammatic way which nervous centers can be removed and the mechanism for walking still be left.

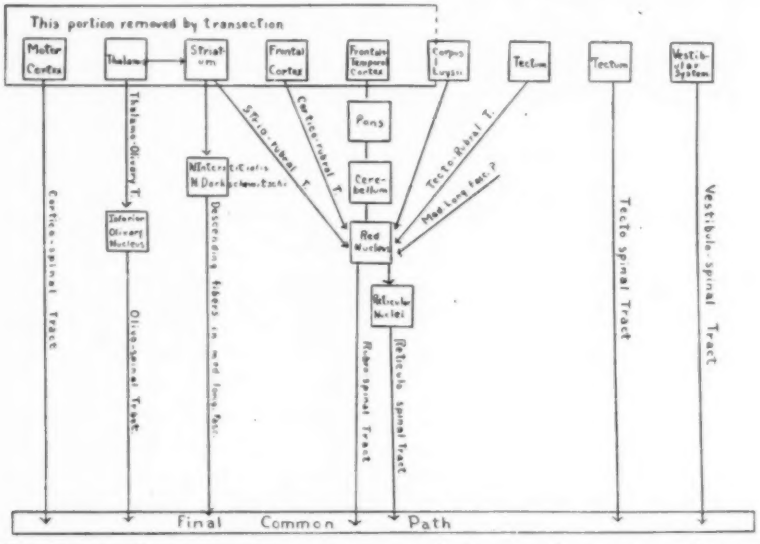

Fig. 3.—A diagram to illustrate some of the prespinal centers that were removed and some that remained in cats which walked.

In our experiments, the complete cerebral cortex was extirpated without destroying the essential nervous pattern for righting and walking. The cerebral cortex exerts both an excitatory and an inhibitory influence on the final common path. The thalamus (with the exception of the most caudal tip) and all of the neostriatum and paleostriatum were removed, so that they cannot be considered as necessary in the mechanism for walking. The absence of these structures may explain the distortions in tonus that have been described for certain postures.

There are a number of mechanisms remaining which need to be considered as involved in reflex walking. The red nucleus, according to Magnus and Rademaker, is a center for a number of the righting

47. Langworthy, O. R.: Physiol. Rev. **8**:151, 1928.

reflexes and for distribution of tonus. The tectum and its projection system needs to be considered. The vestibular system with its descending pathways, the vestibulospinal tracts, and its ascending pathways in the medial longitudinal fasciculus is probably essential. The work of Muskens [48] suggested the nucleus interstitialis and the nucleus darkschewitschi and their descending pathways in the medial longitudinal fasciculus as possibilities. In the present state of knowledge, it is difficult to say whether the substantia nigra and the cerebellum are necessary for the nervous pathway of walking. The function of the cerebellum in relation to the mechanism for walking can best be studied in animals in which the cerebral hemispheres have been removed in addition to decerebellation.[49] The work of Mella [24] and of Morgan [50] pointed to the subthalamic nucleus of Luys as a part of the pathway. At least two thirds of this structure was present in all cats that walked. In addition to the prespinal mechanisms that have been mentioned, we should include the proprioceptive impulses arising in the neck muscles supplied by the upper cervical segments, which act on a center situated caudal to the midbrain to produce neck righting reflexes and on a center on the rostral portion of the cervical cord to produce tonic neck reflexes. According to Graham Brown,[42] there is in the spinal cord itself an innate mechanism responsible for rhythmicity. The afferent impulses of exteroceptive, interoceptive and proprioceptive origin from the body may act directly through a spinal pathway on the neurons of the final common path or they may act indirectly by passing up the spinal cord to reach one of the prespinal centers, i. e., the body reflexes acting on the body pass to a prespinal center in the mesencephalic tegmentum and are then relayed down the cord.

In the adult cat, the mechanisms responsible for rhythmicity, static or postural tonus and equilibration and ability to maintain an upright posture are coordinated into the complete act of walking by nerve centers and pathways found caudal to a plane of section passing from the rostral border of the superior colliculi through the rostral portion of the mammillary bodies. This has been found by Windle [51] to be the case in newly born and young kittens and puppies. He also found that sections at a lower level were followed by a condition of decerebrate rigidity and a loss of the complete act of walking. His observations in young animals are similar in every way to those in adult animals which we have described. It has been pointed out that experiments with monkeys have been unsatisfactory and inconclusive. It is doubtful that man with a similar amount of intact central nervous system would

48. Muskens, L. J. J.: Brain **45**:454, 1922; J. Physiol. **64**:303, 1928.

49. Rademaker, G. G. J., and Winkler, C.: Versl. d. k. Akad. v. Wetensch. Wis-en natuurk. Afd., Amsterdam **37**:356, 1928.

50. Morgan, L. O.: J. Comp. Neurol. **44**:379, 1927.

51. Windle, W. F.: J. Comp. Neurol. **48**:227, 1929.

present a similar walking pattern. We believe that this would be due to the fact that in the central nervous system of man there has been a migration of function corticalward. The cerebral cortex has become a dominating factor in the physiology of the central nervous system and it may well be that some subcortical centers, i. e., the corpus striatum, the hypothalamic nuclei and the substantia nigra, have a different rôle to play in the complexities of neurologic behavior.

Experiments in which the cerebral cortex with different portions of the brain stem has been removed, have been criticized on the ground that one cannot draw any conclusions as to normal function when just a portion of the central nervous system is present. However, we do know from experiments of this kind how much of the central nervous system we can remove and still leave certain neurologic patterns. Any one of the reflex patterns needs to be considered as being subserved by a number of different mechanisms in the central nervous system which under normal conditions are in equilibrium. Certain of these mechanisms can be removed and this equilibrium may still be maintained, but after extirpation of additional ones, the equilibrium may be so disturbed that some one or other of the reflex patterns predominates. We can remove whatever inhibitory influence the cerebral cortex and the lentiform nucleus may have and still find normal tonus in the animal in some postures. We can remove almost all of the thalamus, leaving only a portion of the hypothalamus, and a similar condition persists, with equilibrium being maintained fairly well. However, when the hypothalamus is removed in addition to the other structures mentioned, the equilibrium in all postures is completely overthrown, the inhibitory forces have lost the battle and the reflex pattern of decerebrate rigidity takes over the situation. We cannot think of the inhibition of hypertonus and decerebrate rigidity as being found in one center or mechanism and there only; but we need to think that it may be found in several mechanisms which under normal conditions are properly balanced and equilibrated to produce normal distribution of tonus in all postures.

CONCLUSION

By making sections at different levels through the brain stem of the adult cat, we found that the most caudal plane of section that could be made and the walking pattern still be left intact was one extending from the rostral border of the superior colliculi to the rostral portion of the mammillary bodies. This method gave exact information as to what structures were removed. Some of the prespinal centers which remained and should be considered as possibly involved in walking and the regulation of tonus were: the red nucleus and the hypothalamic centers remaining, the substantia reticularis, the tectum, the cerebellum and the vestibular system. It is impossible for one to say which ones of these remaining structures are utilized, but some-

where in the complexity of these nervous centers and pathways must be located the mechanisms controlling the complete reflex act of walking. The upper part of the mesencephalic tegmentum and possibly its continuation into the hypothalamus are necessary for the regulation of tonus and the maintenance of equilibrium, which make locomotion possible.

PROTOCOLS OF EXPERIMENTS

CAT 12.—On June 22, 1928, at 10:30, the operation was concluded. The subsequent observations were as follows:

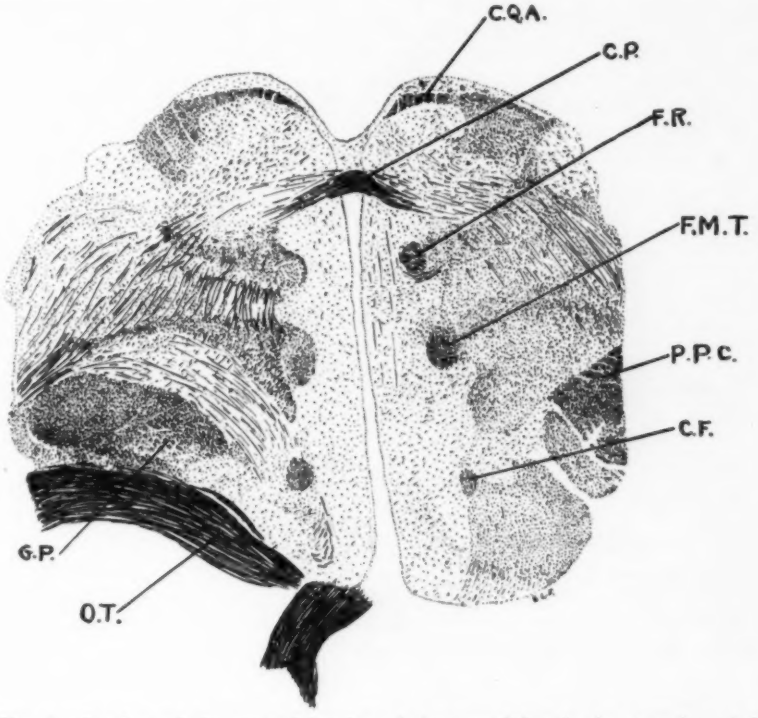

Fig. 4.—Section of the rostral border of the remaining brain stem in cat 12. The abbreviations indicate: *C.Q.A.*, corpus quadrigeminum anterius; *C.P.*, commissura posterior; *F.R.*, fasciculus retroflexus; *F.M.T.*, tractus mammillothalamicus; *P.P.C.*, pes pedunculi cerebri; *C.F.*, columna fornicis descendens; *G.P.*, globus pallidus; *O.T.*, tractus opticus.

10:45: The cat was lying on the right side, the hind limbs rigidly extended and the front limbs held with the humerus against the chest and the elbow extended. There was a definite tendency on the part of the animal to right itself.

10:50: The animal was on its back; the head and shoulders were flexed; it raised the forepart of the body and rolled over to the right in an effort to get up. With the animal on its back, there were definite lengthening and shortening reactions in the hind limbs. The rigidity in the fore limbs was variable, with a continuous extensor rigidity at the elbow; the upper part of the arm was

held pressed against the chest. With the animal on the right side, the front and rear right limbs were more rigid than the left. With the animal on the left side, the rigidity was equal on both sides.

11:00: The cat walked well on the hind limbs, but had difficulty in getting the fore limbs out in front because of holding the upper arm against the chest. It righted itself and walked on hind paws and on wrists.

11:17: It walked along with nearly perfect coordination, but still walked on wrists.

12:55: It walked perfectly on both fore and hind paws.

1:07-3:57: It showed spontaneous periodic righting and walking.

4:00: The cat spontaneously righted itself, walked across the room, butted its head against the wall, then turned and walked back again with normal posture in both fore and hind limbs. The animal did not fall and the gait was normal. This was a distance of 15 feet (457.4 cm.) each way.

4:09-5:21: It showed periodic spontaneous righting and walking.

5:28: It climbed 3½ feet (106.68 cm.) well. It walked normally.

5:30: The animal was killed.

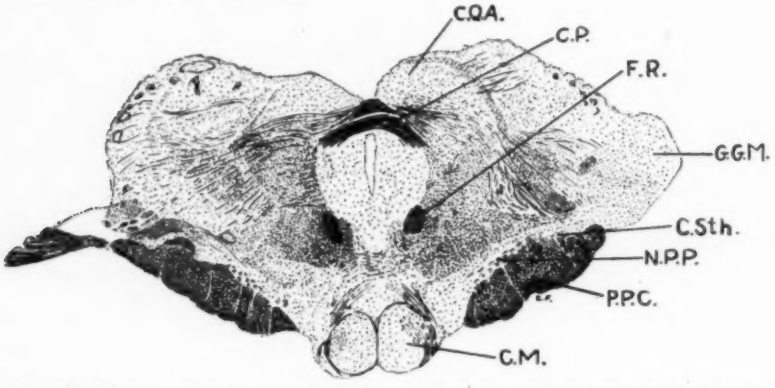

Fig. 5.—Section of the rostral border of the remaining brain stem in cat 17. *C.Q.A.*, corpus quadrigeminum anterius; *C.P.*, commissura posterior; *F.R.*, fasciculus retroflexus; *G.G.M.*, ganglion geniculatum mediale; *C.Sth.*, corpus subthalamicum; *N.P.P.*, nucleus proprius pedunculi cerebri; *P.P.C.*, pes pedunculi cerebri; *C.M.*, corpus mammillare.

Artificial heat had been applied to the cat to maintain its normal body temperature.

Gross Examination.—The section passed from the pineal body to just in front of the optic chiasm.

Microscopic Examination.—The pallidum was present right and left. The red nucleus, the corpus subthalamicum and the nucleus proprius pedunculi were intact.

Cat 17.—On July 2, 1928, at 1:05, the transection was completed. The subsequent observations were as follows:

1:30: The cat showed ineffectual righting reflexes when lying on either side, but it raised head and neck and tried to pull itself up with the fore limb that was uppermost.

4:15: The righting reflex was well developed, and the animal took six or eight steps, but it walked on the wrists and not on the forepaws.

5:15: It walked 7 feet (213.4 cm.) on the wrists and the hind paws.

5:30: It walked spontaneously 10 feet (304.8 cm.).

July 3, 8:50 a. m.: The cat remained alive all night but was stiff when examined.

10:00: The cat died.

This animal failed to maintain a normal body temperature.

Gross Examination.—The section passed from the rostral border of the superior colliculi through the rostral border of the mammillary bodies.

Microscopic Examination.—The pallidum was absent. The red nucleus was intact. The lower two thirds of the corpus subthalamicum and the lower two thirds of the nucleus proprius pedunculi were present.

CAT 20.—On Nov. 5, 1928, at 10:15, the operation was completed. The subsequent observations were as follows:

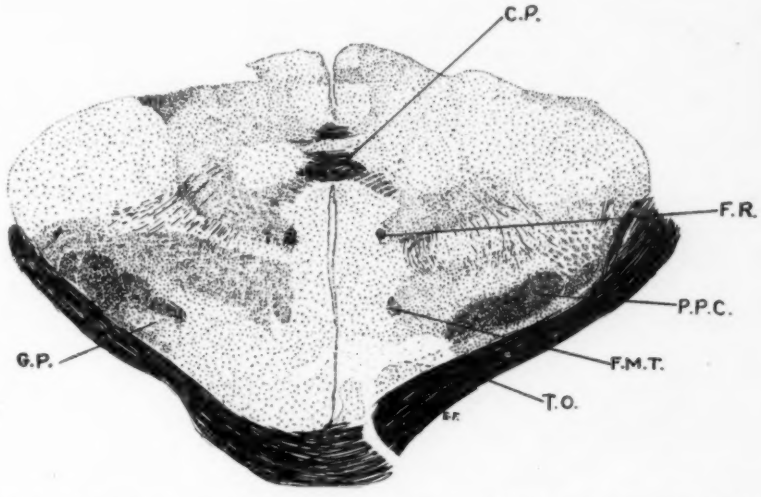

Fig. 6.—Section of the rostral border of the remaining brain stem in cat 20. *C.P.*, commissura posterior; *F.R.*, fasciculus retroflexus; *P.P.C.*, pes pedunculi cerebri; *F.M.T.*, tractus mammillothalamicus; *T.O.*, tractus opticus; *G.P.*, globus pallidus.

10:30: The cat was active and showed progressive movements.

10:58: The cat walked spontaneously ten steps and fell to the right.

11:10: It walked well for some distances. When it approached the wall, it stopped and remained standing until finally the neck reflexes caused a turning in one direction or the other. When the animal was on its right side, it showed a nearly normal distribution of tonus.

11:25-12:47: It had periods of spontaneous righting and walking.

3:10: When placed on its feet, it stood erect a few moments and then walked on all four feet for eight steps.

4:17: It walked spontaneously for 10 feet (304.8 cm.), and climbed up about 4 feet (122 cm.) among the pipes in the corner of the laboratory. It could right itself from either side and was distinctly improved.

4:45: The cat walked spontaneously for long distances. During and following the periods of activity, the respirations were accelerated and deepened.

5:35: It still walked well and could right itself from either side.

5:45: It was still active when put in a warm cage to stay over night. It was found dead the next morning.

This animal maintained its body temperature at about 38 C. throughout the experiment till 5:30, when the last record was taken.

Gross Examination.—The plane of section extended from just in front of the superior colliculus to about the level of the optic chiasm. The optic chiasm was intact, but the plane of section dipped down behind it so that most of the section was at the level of the lower border of the chiasm.

Microscopic Examination.—The pallidum, the red nucleus, the corpus subthalamicum and the nucleus proprius pedunculi were intact.

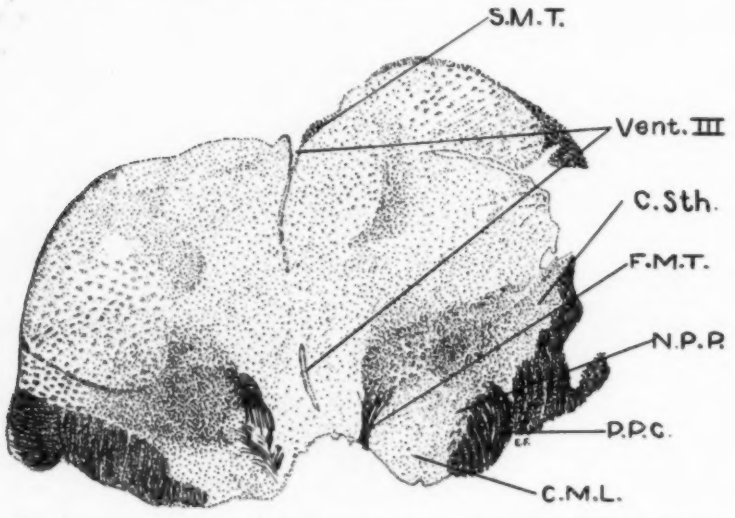

Fig. 7.—Section of the rostral border of the remaining brain stem in cat 31. *S.M.T.*, stria medullaris thalami; *Vent.III*, ventriculus III; *C.Sth.*, corpus subthalamicum; *F.M.T.*, tractus mammillothalamicus; *N.P.P.*, nucleus proprius pedunculi cerebri; *P.P.C.*, pes pedunculi cerebri; *C.M.L.*, corpus mamillare laterale.

Cat 31.—The preparation of cat 31 was completed on Nov. 26, 1928.

10:45: The hind legs showed waves of hypertonicity and the fore legs were flexed at the shoulder. There was not so much hypertonicity in the fore legs as was presented in the hind limbs. No spontaneous movements were observed as the animal was left tied in the ventral position on the animal board.

1:00: The cat maintained a standing position for several minutes but did not walk spontaneously.

1:45: It stood normally for two minutes and took two steps in a well coordinated fashion.

2:10: It showed the righting reflex and walked seven steps, some on the wrists, some on the forepaws.

2:40: When placed on its back, the animal righted itself and walked normally. It stood in an upright position for five minutes without falling. It did

not walk spontaneously from this position, but when placed on its side, it righted itself, and walked.

3:50: The cat could still right itself and walk well. It did not respond to painful stimuli. It did not show the spontaneity which was so evident in animals following some of the other preparations.

4:37: It righted itself and walked, using the fore limbs normally.

4:45: The cat was killed.

The normal body temperature had been maintained.

Gross Examination.—The section passed from 5 mm. in front of the habenular commissure to the rostral portion of the mammillary bodies.

Microscopic Examination.—The pallidum was absent. The red nucleus was intact. The corpus subthalamicum was intact (perhaps just upper end gone). The section passed through the upper end of the nucleus proprius pedunculi.

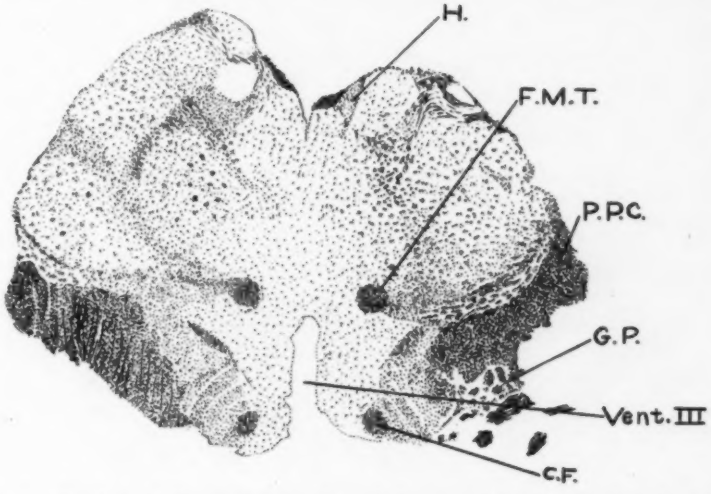

Fig. 8.—Section of the rostral border of the remaining brain stem in cat 32. *H.*, habenula; *F.M.T.*, tractus mammillothalamicus; *P.P.C.*, pes pedunculi cerebri; *G.P.*, globus pallidus; *Vent.III*, ventriculus III; *C.F.*, columna fornicis descendens.

Cat 32.—The animal was brought into laboratory on November 27 and was not fed. On Nov. 28, 1928, at 10:03, the operation was concluded.

10:25: The cat was removed from the animal board. It showed spontaneous righting and walking. The breathing was labored during periods of activity.

10:28-10:40: There was periodic spontaneous righting and walking. In the periods of rest, the fore legs were stiff and showed resistance to passive flexion.

10:42: The cat showed evidences on the face of sham rage. Respirations were labored just before, during and following the periods of activity.

10:48: No hypertonicity was evidenced in any of the legs while the cat was in the crouching position. The respirations were of rapid succession and fairly deep.

11:00-4:00: There was spontaneous periodic righting and walking. The limbs were not spastic in the intervals between walking. The tail was bushy during the periods of activity.

4:15: The cat was killed.

It had maintained its body temperature at 38 C. throughout, in spite of the fact that the room temperature was decreased to 17 C. for about one hour.

Gross Examination.—The section passed from 7 mm. in front of the habenular commissure to the mammillary bodies.

Microscopic Examination.—The pallidum was present on one side. The red nucleus, the corpus subthalamicum and the nucleus proprius pedunculi were intact.

Cat 34.—On Dec. 6, 1928, at 10:30, the operation was completed.

11:00: The cat was removed from the animal board and placed in the hammock. It was extremely active and was difficult to hold until it was placed in the hammock, where it showed rapid running movements. These periods of running movements were followed by periods of rigidity in the limbs.

12:50: The cat was removed from the hammock. It crawled along on its wrists with a staggering gait. When it lay on its left side, it showed some

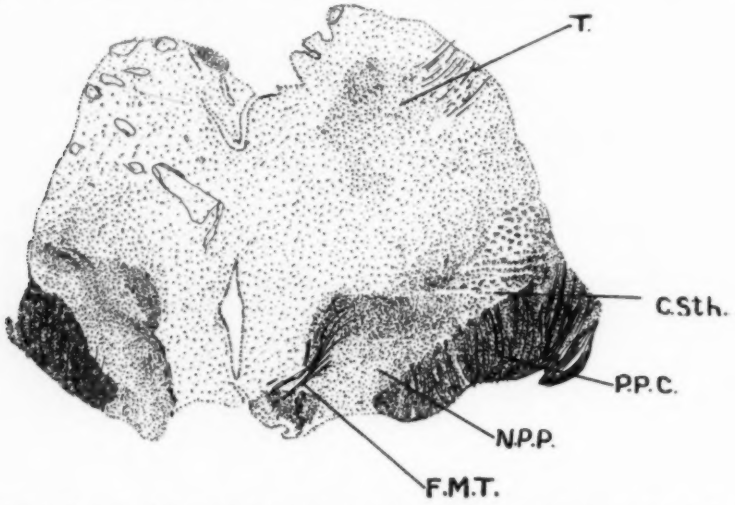

Fig. 9.—Section of the rostral border of the remaining brain stem in cat 34. *T.*, thalamus; *C.Sth.*, corpus subthalamicum; *P.P.C.*, pes pedunculi cerebri; *N.P.P.*, nucleus proprius pedunculi cerebri; *F.M.T.*, tractus mammillothalamicus.

increase of extensor tonus in the hind limbs and in the fore limbs increased flexor tonus. When on its right side, the cat showed hypertonicity of the extensors in the hind limbs with a slight predominance of the flexor tonus in the fore limbs. Sometimes it walked with normal posture and used the forepaws in normal fashion.

12:57: It spontaneously righted itself and walked five steps in normal posture and with normal function of its limbs.

1:01: The cat was in the hammock again. The limbs were held in a posture of decerebrate rigidity with marked resistance to passive flexion in the hind limbs. A slight but definite resistance to passive flexion was observed in the fore limbs.

4:43: The cat seemed to be in the same condition as it had been all day. Spontaneous well coordinated running movements occurred while it was in the hammock.

5:00: The animal was killed.

The normal body temperature had not been maintained, and an electric heating pad had been applied during the experiment.

Gross Examination.—The section passed from 4 mm. in front of the habenular commissure through the rostral part of the mammillary bodies. Lateral cuts did not completely remove the rhinencephalon on either side.

Microscopic Examination.—The pallidum was absent. The red nucleus was intact. The lower two thirds of the corpus subthalamicum and the lower two thirds of the nucleus proprius pedunculi were present.

CAT 40.—On Dec. 8, 1928, at 10:40, the operation was completed.

11:05: The cat was removed from the animal board. Hypertonicity of the extensors was seen in all the limbs. Touching the right hind paw caused fairly well coordinated running movements. The respirations succeeded each other rapidly, 92 per minute, and were shallow.

11:10: The cat was placed in the hammock; the limbs showed the typical posture of decerebrate rigidity.

11:15: The cat, while still in the hammock, showed spontaneous running movements in all four limbs, but the movements in front were not coordinated with those behind.

2:17: It showed the righting reaction and took four steps well, but the upright posture was relatively unstable.

2:21: The animal spontaneously righted itself, walked five steps in normal posture, then stood erect for five seconds and then took six more steps, finally walking off the table.

2:30: It walked normally after righting itself. When it was in the hammock there was hypertonicity, slight in the fore limbs but in the hind limbs so predominant that one could lift the cat by upward pressure against the pads of the hind feet.

2:30-3:25: The cat was placed in the hammock; there were periodic spontaneous running movements.

3:26: While the cat was in the hammock there was resistance to passive flexion in all limbs, more marked in the hind than in the front limbs.

3:32: The cat on its back in the cradle showed hypertonicity of the extensors in the hind limbs, which were plastic. There was a resistance to passive flexion in the fore limbs, but there was a weakness of the extensors of the wrists. There was a predominance of flexor tonus at the shoulder joint. The hind limbs tended to be adducted and sometimes were crossed.

3:35: When the animal was removed from the cradle, it righted itself and walked normally.

4:00: The cat was killed and examined.

The body temperature had been normally maintained.

Gross Examination.—Autopsy revealed a thalamic animal with the plane of section extending dorsally just in front of the rostral tip of the third ventricle and ventrally 1 mm. in front of the optic chiasm. A small portion of the pyriform lobe was present on each side.

CAT 43.—On Dec. 10, 1928, at 10:40, the operation was completed.

11:10: The cat was placed in the hammock; there was a predominance of extensor tonus in all limbs.

11:55: The cat was removed from the hammock; it did not stand but would sit if passively placed in position. While it was resting on its belly, its hind

legs were hyperextended, and there was resistance to extension at the shoulder and to flexion at the elbow. It was possible to mold the limbs into a position normally held by the animal; i. e., the limbs showed the lengthening and shortening reactions and were plastic. There was no evidence of spontaneity. The cat did not step, did not respond to strong pinching of the tail or of the side of the abdomen. There was no opisthotonos. With the animal on its right side, on its left side and on its back, the reactions were those typical of decerebrate rigidity.

12:50-3:30: During this period, there were periodic spontaneous running movements which were coordinated in the fore limbs, but the hind limbs moved back and forth together. The animal was given every opportunity to walk, but the righting reactions were incompletely developed, and the animal never was able to walk. With the cat on the table, these spontaneous running movements occurred, but the animal never righted itself.

3:30: Up to this time, the normal body temperature was maintained. The cat was taken outside, where the temperature was 7 C. and in one hour and a half the rectal temperature fell to 33 C. The spontaneous movements no longer occurred.

5:05: The animal was killed.

Gross Examination.—The section passed from the rostral border of the superior colliculi to just rostral to the point of exit of the oculomotor nerves.

Microscopic Examination.—The pallidum was absent. The lower half of the red nucleus was intact. The corpus subthalamicum and the nucleus proprius pedunculi were absent.

Rabbit 38.—On Dec. 11, 1928, a rabbit was decerebrated for thalamic preparation. The operation was completed at 11:45. The subsequent observations were as follows:

12:55: With the animal in the hammock, there was a definite resistance to passive flexion which was greater in the hind limbs than in the fore limbs.

1:55: Removed from the hammock and placed on the table in a normal crouching position, the rabbit supported itself and licked its forepaws. It maintained its upright posture even against strong pushing; one could hardly push it over. It righted itself when its normal posture was disturbed. There were no spontaneous hopping movements. When placed on its side or back, it did not right itself spontaneously, but did if its toe was pinched. It was stimulated into hopping when the toes of the right hind leg were pinched. During progression it hopped just like a normal rabbit and maintained its normal equilibrium. Pinching the skin of the back and thigh did not produce any reaction. Pinching the toes of the fore limbs caused them to be withdrawn, but the animal did not hop.

3:40: Extensor tonus still predominated while the rabbit was in the hammock, more marked in the hind than in the front limbs.

5:13: With the animal on the table, pinching the tail caused it to hop actively. It still maintained its equilibrium well.

December 12, 8:53: It walked and jumped on stimulation and maintained its equilibrium. While it was in the hammock, it still showed resistance to passive flexion in all limbs, more marked in the hind than in the front limbs.

9:30: The animal was killed. The temperature had been well maintained during the first ten hours of the experiment, but it had fallen during the night.

Gross Examination.—The animal showed a thalamus preparation with the section passing in front of the optic chiasm.

ABSTRACT OF DISCUSSION

DR. WILDER PENFIELD, Montreal: This work of Dr. Ranson's really opens up a new horizon in the study of the effects of removal of the brain. Goltz, with his well known dogs, demonstrated that an animal, the brain of which has been removed down to the thalamus, is capable of walking, of running and of taking its food. Sherrington showed that decerebration through the midbrain was followed by decerebrate rigidity or standing rigidity. Subsequently, a great deal of work has been done on the analysis of the various components of the condition which follows decerebration.

We are all familiar with the work of Magnus and de Kleijn which showed that after a low decerebration the righting reflexes are absent and they become more and more complete as the level of decerebration is carried upward in the midbrain, so that when the upper end of the red nucleus is intact, the animal is capable of righting itself, and shows various neck and body reflexes.

This work of Dr. Ranson carries the section just a little higher. It is amazing to find that section only a distance of about 1 cm. farther forward in the ventral surface, and no farther forward on the dorsal surface, produces an animal that has a number of functions which the decerebrate animal has not, an animal which has temperature control, an animal which is capable of normal walking. To any one who has studied and observed decerebrate animals, it is obvious that these animals are capable of something in the way of locomotion of which a decerebrate animal is not capable.

I have noticed that in an anterior decerebration, that is, one which goes through the top of the midbrain, the animal is often excited, jumps, may kick, and may show running movements, but it lacks the ability of coordinated walking. Therefore, the centers, or the completion of the reflex arcs, which are necessary for the integration of the components of walking must lie, in the cat at any rate, in that small slice.

It is possible, in addition, that there are sympathetic centers in this portion of the brain which Dr. Ranson has spared. The corpus subthalamicum of Luys is already laden with certain sympathetic functions.

This work seems to me of the utmost importance, but the question that comes to mind is: What application of these observations can one make to human physiology? It is true that one cannot carry over the observations in the cat to man. The step to monkey has not yet been clearly shown, that is, at just what level of brain removal it is still possible to get walking movements. And it is evident that within the brain certain functions tend to migrate upward as the phylogenic scale rises. The rabbit which is decerebrated at a point similar to that used to produce rigidity in the cat does not become a decerebrate preparation. And so, in man, similar reactions may perhaps require higher centers than those pointed out by Dr. Ranson.

DR. RANSON: The little wedge that I showed in the picture is not nearly 1 cm. thick at its thickest part; I should say it is not more than 3 mm. thick; so that it is really a small bit of brain which makes the difference between a rigid decerebrate preparation and a walking hypothalamic cat.

We were not able to satisfy ourselves as to just how much regulation of temperature was left in the hypothalamic cat. Certainly, it was not so perfect as in the thalamic cat, but our data are not sufficient on this point.

I agree with Dr. Penfield that these results cannot be carried over directly to man. No one has yet succeeded in obtaining a walking thalamic monkey.

THE PACCHIONIAN SYSTEM

HISTOLOGIC AND PATHOLOGIC CHANGES WITH PARTICULAR REFERENCE TO THE IDIOPATHIC AND SYMPTOMATIC CONVULSIVE STATES *

N. W. WINKELMAN, M.D.
AND
TEMPLE FAY, M.D.
PHILADELPHIA

The physiologic emphasis placed on the function of the arachnoid villi and the pacchionian granulations by Weed and his co-workers (1914-1923) has led us to a careful histologic and pathologic study. As the probable point of most active elimination of cerebrospinal fluid, their structure, development and reactions to disease should be of importance in many cases in which gross evidence of increased accumulation of cerebrospinal fluid over the cortex has been observed. These collections of cerebrospinal fluid demonstrated by encephalography, operation and necropsy suggested that the function of the structure concerned in elimination of the cerebrospinal fluid might be deficient.

Weed (1914-1923) demonstrated that the arachnoid villi, present at birth, develop in adult life into the well known pacchionian granules, and by the use of true colloidal solutions demonstrated that the major portion of cerebrospinal fluid finds its outlet through these structures. Weed's conclusions in his paper on "The Absorption of Cerebrospinal Fluid Into the Venous System," [1] are as follows:

> The pathway of absorption of the cerebrospinal fluid into the blood stream under normal conditions is by way of arachnoid villi into the great dural venous sinuses. Under the influence of an increased salt-content of the blood, effected by the intravenous injection of strongly hypertonic solutions, absorption takes place also by way of the perivascular channels and through the ependymal lining of the cerebral ventricles into the capillary bed of the nervous system. In the normal process, filtration may be the physical factor of greatest importance, but after the intravenous injection of strongly hypertonic solutions, osmosis and diffusion apparently play the only active rôles.

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* Submitted for publication, May 1, 1929.

* Read at the Fifty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 28, 1929.

* From the Daniel J. McCarthy and Memorial Foundations, University of Pennsylvania, Philadelphia, and the Laboratory of Neuropathology of the Philadelphia General Hospital.

1. Weed, L. H.: The Absorption of Cerebrospinal Fluid Into the Venous System, Am. J. Anat. **31**:191 (Jan.) 1923.

A review of the literature shows a marked scarcity of observations on the pacchionian bodies and their pathologic changes as related to the various diseases affecting the cerebral hemispheres.

REVIEW OF THE LITERATURE

In 1876, Key and Retzius[2] published their monumental work, "Studien des Anatomie des Nervensystems und des Bindesgewebe," in which a comprehensive description of the cerebral membranes was given for the first time. They demonstrated the continuity of the spinal and cerebral subarachnoid spaces by injection of colored gelatin solutions. The solution passed through the pacchionian granulations into the cerebral sinuses. With silver nitrate stains they demonstrated an endothelial covering over the entire pacchionian granulation. A subdural space was also noted in each pacchionian granulation. By simultaneous injections into the subarachnoid and subdural spaces the pacchionian bodies and the subdural spaces about them could be filled, but no fusion of the injection solutions occurred. A study of their preparations led them to conclude that the cerebrospinal fluid passed normally from the subarachnoid space into the subdural space about the granulation, and from this directly into the sinus. They postulated the view that there were stomas between the covering cells through which this occurred. They thought that it depended on osmosis and differences in pressure between the subarachnoid fluid and the venous sinuses. They also found that a small part of the solution passed along the cerebral nerves into lymphatic vessels.

While this work was accepted for a while, it was soon thought that the pacchionian system could not be the mechanism for the normal escape of cerebrospinal fluid, because these bodies are not fully developed, except after puberty, and they were not supposed to occur in the lower animals. The older theories of Calmeil[3] (1826) and Rokitansky[4] (1844) were therefore revived.

Reiner and Schnitzler[5] (1894) furnished physiologic proof of venous absorption of the cerebrospinal fluid by injection of potassium ferrocyanide in physiologic solution of sodium chloride and also olive oil into the subarachnoid space and by the demonstration of these substances in the jugular vein. Because of the absence of pacchionian

2. Key and Retzius: Anatomie des Nervensystems und des Bindesgewebe, Stockholm, P. A. Norstedt & Söner, 1876.

3. Calmeil, quoted by Key and Retzius: Anatomie des Nervensystems und des Bindesgewebe, Stockholm, P. A. Norstedt & Söner, 1876.

4. Rokitansky, quoted by Key and Retzius: Anatomie des Nervensystem und des Bindesgewebe, Stockholm, P. A. Norstedt & Söner, 1876.

5. Reiner, M., and Schnitzler, J.: Ueber die Abflusswege des Liquor cerebrospinalis, Zentralbl. f. Psychol. **8**:684, 1894.

bodies in their animals, they believed that the method described by Key and Retzius was incorrect.

Leonard Hill [6] (1896) was able to demonstrate solutions of methylene blue (methylthionine chloride, U. S. P.) in the various organs in from ten to twenty minutes by what he thought was a direct path into the venous sinuses, but he could not demonstrate the dye in the lymphatics of the neck within this time.

Lewandowsky [7] (1900) was able to get the chemical reaction for sodium ferrocyanide in the urine of animals within thirty minutes after intraspinal injection.

Cushing's conclusions, in 1902, were that the pacchionian granulations were not actively concerned in the elimination of cerebrospinal fluid because of their absence in children and in laboratory animals. He postulated some valvelike mechanism similar to the emptying of the thoracic duct into the venous circulation.

In 1910, Mott [8] conceived an entirely new mechanism by postulating the passage of spinal fluid through the walls of the cerebral capillaries. In the brains of animals in experimental anemia, he found distinct spaces about the capillaries and about each nerve cell which were proved by chemical and histologic methods to be filled with cerebrospinal fluid.

Goldmann [9] (1913) in vital staining experiments believed that lymphatic drainage was the essential one for cerebrospinal fluid, although he himself sensed the shortcomings of this method.

Dandy and Blackfan [10] (1913) confirmed the observations that venous absorption is greater than along lymphatics, and from the results of their experiments contended that the absorption of cerebrospinal fluid is a diffuse process from the entire subarachnoid space."

In a more recent work, Dandy (1921), on what he termed communicating hydrocephalus, reiterated his belief that the subarachnoid space over the convexity of the brain represents the absorptive area.

The work of Weed [11] (1914-1915) with physiologic investigation of the drainage of the cerebrospinal fluid is of the greatest importance in determining the ultimate pathway, whether venous or lymphatic. He

6. Hill, Leonard: Physiology and Pathology of the Cerebral Circulation, London, J. & A. Churchhill, 1896.

7. Lewandowsky, M.: Zur Lehre von der cerebrospinal Flüssigkeit, Ztschr. f. klin. Med. **40**:480, 1900.

8. Mott, F. W.: The Oliver-Sharpey Lectures on the Cerebrospinal Fluid, Lancet, 1910, p. 1084; July 2 and 9, 1879, part 11.

9. Goldmann, E. E.: Vitalfärbung am Zentralnervensystems, Berlin, G. Reimer, 1913.

10. Dandy, W. E., and Blackfan, K. D.: An Experimental and Clinical Study of Internal Hydrocephalus, J. A. M. A. **61**:2216 (Dec. 20) 1913.

11. Weed, L. H.: Studies on Cerebrospinal Fluid, J. M. Research **26**:21, 51, 93 and 167, 1914-1915.

used what he termed true solutions in contrast to granular suspensions, viscous fluids or emulsions. His use of isotonic ferrocyanide solutions has proved of extreme value in the final elucidation of this important problem. The second point of importance in his technic is the pressure under which the injection is made. Key and Retzius used a pressure of not more than 60 mm. of mercury. The work of those who used excessive pressure cannot be considered. Weed believed that the celebrated observations of Key and Retzius offer but little proof of the function of the pacchionian granulations in the drainage of cerebrospinal fluid, for in their drawings of the passage of the gelatin solution into the sinus, rupture of the wall of the granulation had evidently occurred.

Weed's results are so interesting that they will be summarized in some detail.

Weed [12] stressed the fact that care has to be exercised in the injections since pressure that is too high may cause artificial routes to be opened. He found that fluid from the spinal canal first sought the basilar systems, while that introduced into the cerebellar cisternae or above the tentorium flowed toward the superior longitudinal sinus, and he ended with the significant conclusion, "The chief method of return of cerebrospinal fluid to the general circulation is by a process of filtration through arachnoid villi into the great sinuses."

The question of the presence of the pacchionian granulations in laboratory animals and in children is of importance. Weed found arachnoid villi present in the cat and dog and these are most marked along the great sagittal sinus. This has been our observation also. He also found the same morphologic constituents in the arachnoid villi of infants: [13]

> For while no pacchionian granules are found in infants, these villi are invariably met with in normal children—for undoubtedly, the pacchionian granulation must be considered as a large, hypertrophic villus becoming evident on macroscopic examination in most adults. The more important arachnoid villus, however, is found at all ages in man, but is not of sufficient size to be evident except on microscopic examination.

ANATOMIC CONSIDERATIONS

The pacchionian system is so intimately related to the cerebral membranes that their anatomy should be described. The dura is the outer, dense fibrous envelop which in the cranial cavity is closely adherent to the bones of the skull, but in the spinal canal is supported in a fatty cushion. Weed (1914) expressed the belief that "on its inner surface is a layer of characteristic mesothelial cells, demonstrable by silver nitrate reduction in the intercellular cement substance." Mallory [14] (1920)

12. Weed (footnote 11, p. 91).
13. Weed (footnote 11, p. 62).
14. Mallory, F. B.: J. M. Research **41**:349, 1920.

has been unable to see these lining cells. We have also been unable to demonstrate them. While most textbooks describe two layers in the dura, they are not to be separated and there is rather a splitting to enclose the large venous sinuses.

The arachnoid is a delicate membrane situated between the pia and the dura, and follows the latter rather closely, being separated from it by a definite space known as the subdural space which contains a clear, yellowish fluid (Penfield,[15] 1924). It passes over the various eminences and depressions on the cerebrum and cerebellum without dipping down into the sulci and smaller grooves. It is a membrane without blood vessels. Microscopically, it is made up of mesothelial cells. It is really the outer membrane of a water-tight sack that encloses the entire brain and spinal cord, which at no point allows a communication with the overlying subdural space.

The pia is a delicate, fibrous and highly vascular membrane which immediately invests the brain and spinal cord. It dips down into all the sulci and fissures and lines them to their depths. It is connected with the arachnoid by fine strands of tissue which traverse the space between the pia and arachnoid, or the subarachnoid space, in which the spinal fluid is contained.

The Pacchionian Granulations.—Closely connected with the subarachnoid space are numerous cauliflower-like excrescences of the arachnoid, for the most part small but occasionally more than 0.5 cm. in diameter, which lie along the course of the dural venous sinuses (fig. 2). While most numerous on either side of the superior longitudinal sinus, where they occur in groups, they occur in smaller number and size in connection with the other sinuses. These are the so-called pacchionian granulations. They are invaginated into spaces within the dura known as the lateral lacunae or blood lakes which, according to our investigations, do not contain blood. A few are in relation to the superior sinus itself. As invaginations they are in relation to the subarachnoid space and really are prolongations of the pia-arachnoid or the subarachnoid space (fig. 3). The larger granulations form indentations on the inner table of the skull which can be easily seen in the encephalogram. They increase in size and number as age advances. They are absent in the child, their place and function being taken over by the arachnoid villi as shown so well by Weed. They communicate with the subarachnoid space through small stomas and are consequently filled with cerebrospinal fluid.

The microscopic structure of the pacchionian granule is simple. There is a central cavity containing a fine reticulum exactly similar to

15. Penfield, W. G.: The Cranial Subdural Space (A Method of Study), Anat. Rec. **28**:173, 1924.

the reticulum present within the subarachnoid space (fig. 4). During life this is filled with spinal fluid and after death shows as a myxomatous material (fig. 5) which under high magnification is seen to be granular.

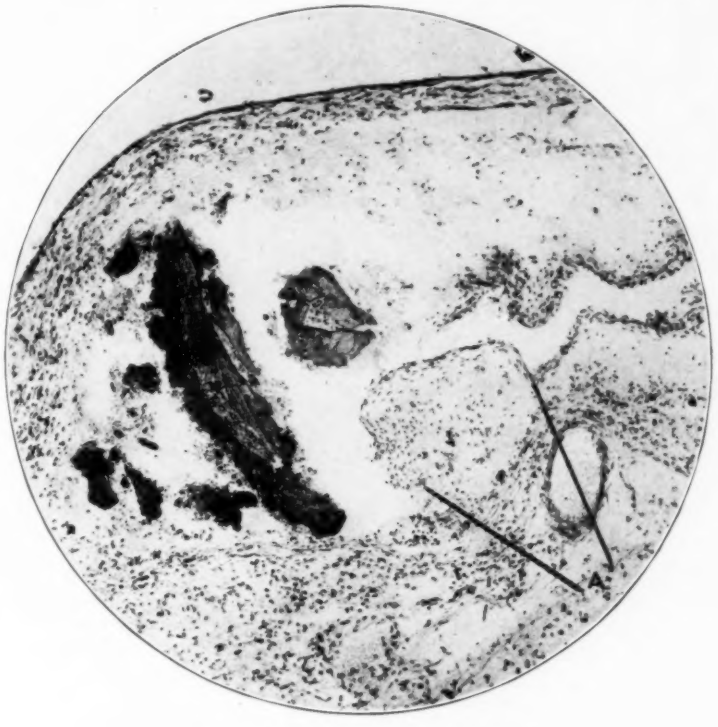

Fig. 1.—Normal arachnoid villus.

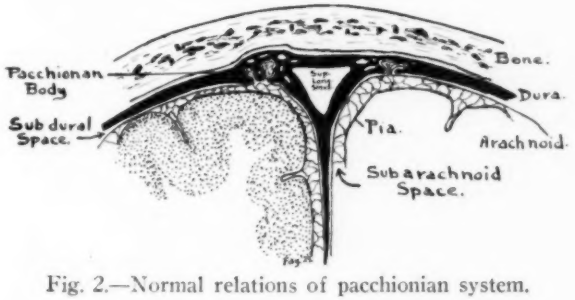

Fig. 2.—Normal relations of pacchionian system.

The arachnoid is its immediate covering in the same way that it encloses the subarachnoid space. This arachnoidal membrane may consist of only a single layer of cells, but, as age advances, thickening of this membrane occurs here as it does in the general arachnoid of the subarachnoid

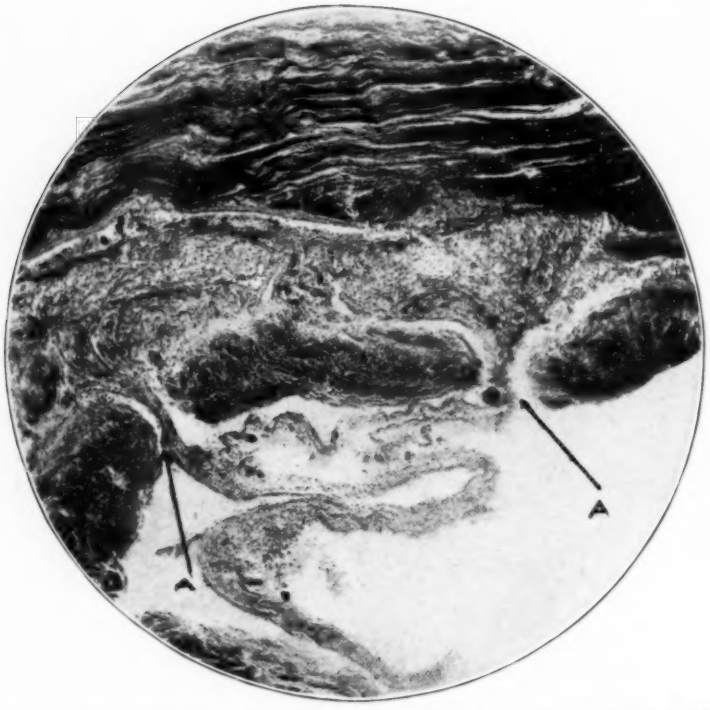

Fig. 3.—Two normal pacchionian bodies, *A*, projecting into the dural envelop.

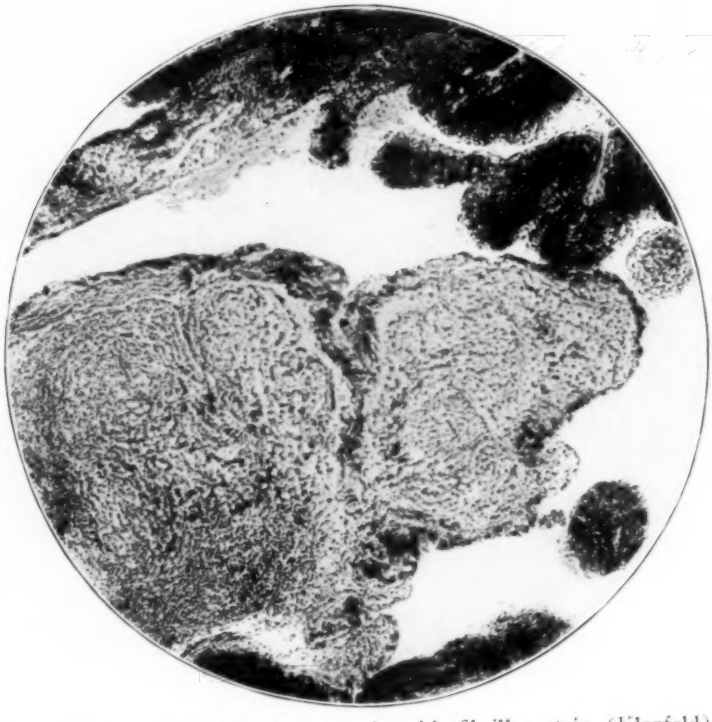

Fig. 4.—Normal pacchionian granule with fibrillar stain (Klarfeld).

space, so that eventually the membrane may appear from ten to fifteen layers of cells thick (fig. 6).

While there is no more space between the arachnoid and the dura within the pacchionian granule than there is in the general cranial cavity, still the dural covering is not in any more intimate relation here than elsewhere throughout the brain. An actual subdural space here was denied by Weed but was found by Key and Retzius. We have seen such a space under abnormal conditions, and it is well known that the

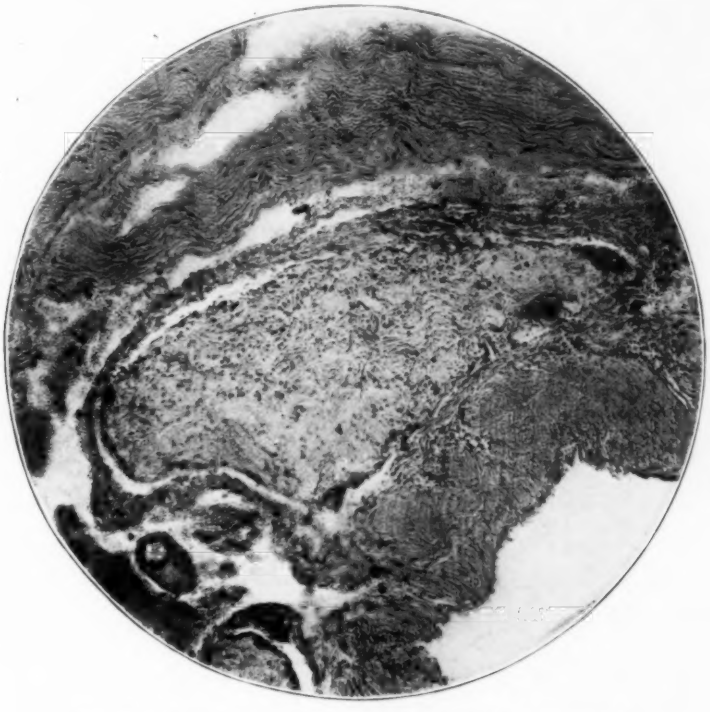

Fig. 5.—Myxomatous material within pacchionian nodule.

subdural space within the general cranial cavity can increase in size under abnormal conditions also, as when subdural hemorrhage or fluid accumulations occur.

As already mentioned, the pacchionian granules project into what are known as the lateral lacunae or blood lakes, as a rule, and not directly into the superior sinus. These lacunae are in communication with and eventually lead into the spaces surrounding the superior sinus in the same manner as the spaces within a sponge communicate with one another (fig. 7). At no time have we found blood within these so-called blood lakes, but they are undoubtedly filled during life with

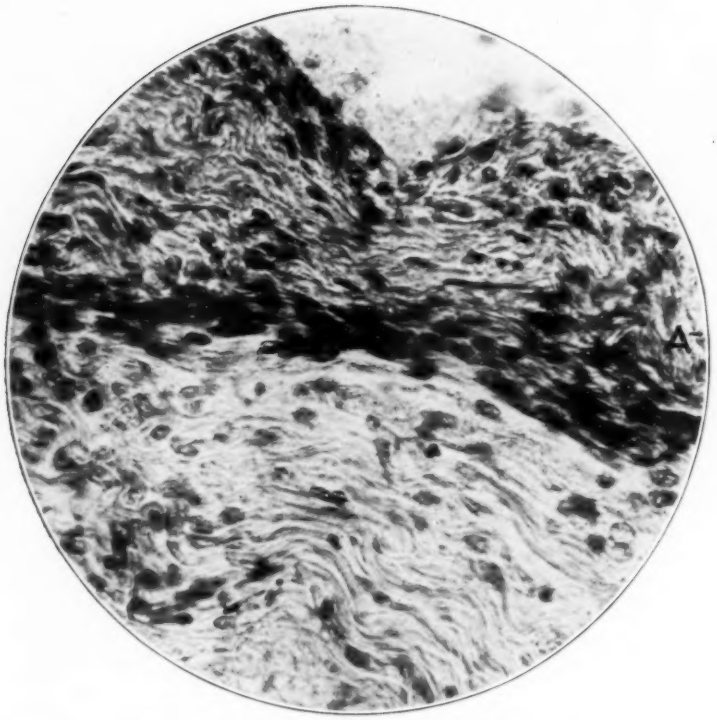

Fig. 6.—Thickened arachnoid covering, *A*.

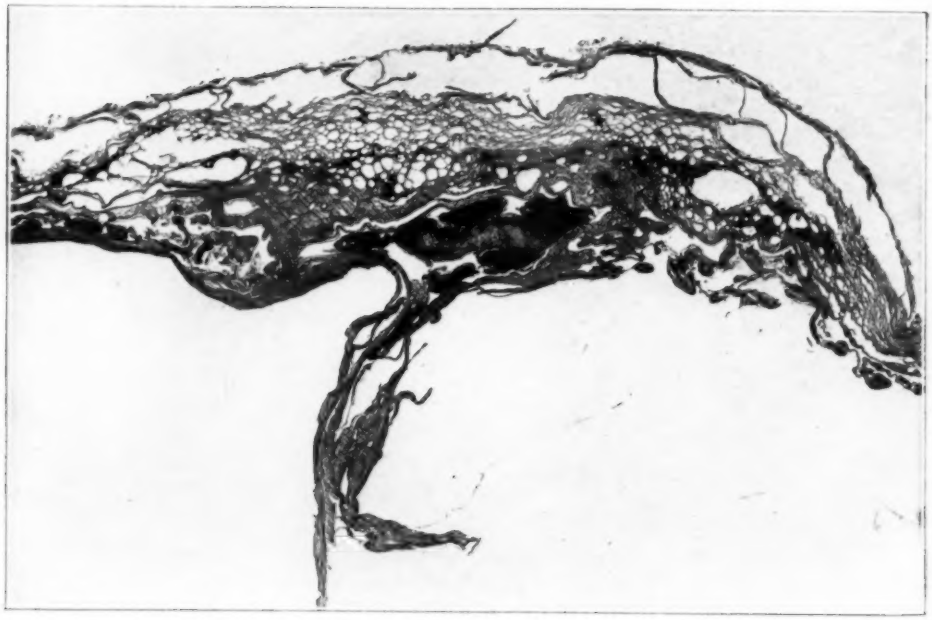

Fig. 7.—Relation of the lateral lacunae to the superior sinus.

spinal fluid on its way to the superior sinus. Cushing [16] (1914) made mention of the fact that puncture here by the surgeon produces the oozing of a thin watery secretion. Our conception of the mechanism of spinal fluid elimination is through the pacchionian granulations into the lacunae and then as a result of dialysis and pressure into the great venous sinuses.

DEVELOPMENT OF THE PACCHIONIAN GRANULATIONS

It is admitted by all who have worked on this subject that the characteristic granulations along the superior sinus occur only in the adult. Differences of opinion as to the mechanism of drainage of the cerebrospinal fluid occur because of the absence of these bodies in children and in the ordinary laboratory animals. The embryologic studies by Weed have shown rather conclusively that the predecessor of the pacchionian granule is the arachnoid villus which is found in children and in animals; the physiologic importance is the same. It is only as age advances that the arachnoid villi develop into the pacchionian granules of the adult.

PATHOLOGIC OBSERVATIONS IN THE PACCHIONIAN SYSTEM

As a result of the study of the pacchionian granulations in a series of more than 200 cases, from all varieties of clinical and pathologic conditions, we have found that we can subdivide the conditions encountered into definite and distinct groups. Borderline cases occur as in every other attempt at classification, but they are in the great minority.

Group 1.—*Aplasia.*—In this group we have placed those cases, fourteen in all, in which, even at times with practically serial section study of the dura in relation to the superior longitudinal sinus, no fully matured pacchionian granules were found. It is true that when sections of the upper portions of the hemispheres were made along with the attached dura, we did find arachnoid villi, but at times even they were less numerous than in most of the children's brains. This condition is probably to be considered a developmental fault and has been observed by Cushing [17] "in a few cases of essential hydrocephalus" which he has had the opportunity of studying. In going over our records of the cases in which this condition occurred we found that it was mainly in so-called idiopathic epilepsy (eight cases) and several cases of hydrocephalus of unknown etiology. It might be mentioned in passing that with this condition in epileptic brains there has also been associated an infantile appearance of the entire brain. While the amount of pacchionian granules shows considerable variation in the different cases, it

16. Cushing, H.: Studies on the Cerebrospinal Fluid, J. M. Research **26**:1, 1914-1915.

17. Cushing (footnote 16, p. 8).

is comparatively rare in the adult to meet with a case in which there are practically no granulations along the lateral margins of the superior sinus. We have chosen to call this absence of the pacchionian granules an aplasia of the pacchionian system, representing as it probably does a defective development.

Group 2.—*Hypoplasias.*—In these cases, while there is a development of the pacchionian system that development has not reached full maturity.

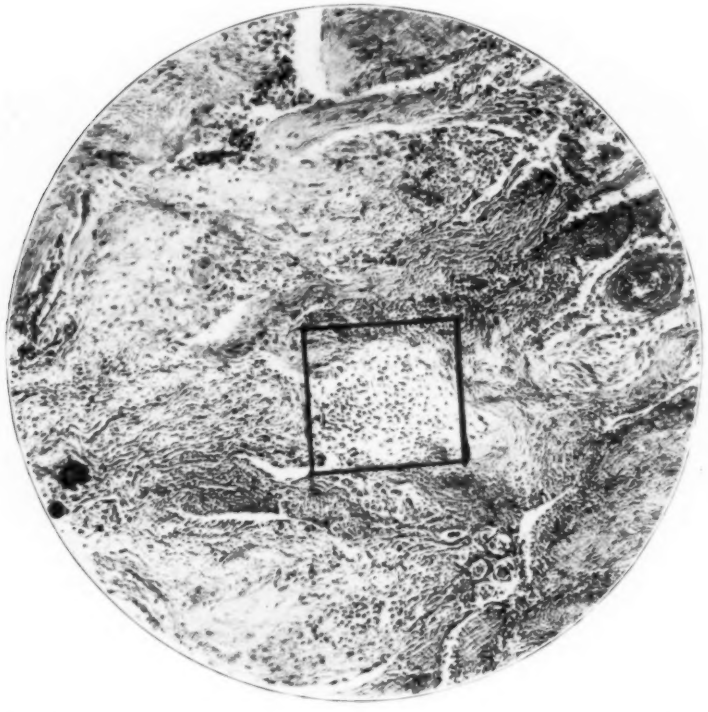

Fig. 8.—Atrophic masses of mesothelial cells within the dura, representing poorly developed pacchionian granulations.

As one examines section after section of the superior sinus and its environs, there are encountered groups of mesothelial cells within the meshes of the dura (figs. 8 and 9), but there is no normal development into the adult forms of granules and they show no evidences of functional activity. Bagley [18] (1927) has found that as a result of hemorrhage into the subarachnoid space there may be narrowing or even occlusion of the stomas of the pacchionian granulations with resultant atrophy.

18. Bagley, C.: Functional Disturbances and Organic Alterations Following the Introduction of Blood into the Cerebrospinal Fluid, Paper read before the Association for Research in Nervous & Mental Disease, Dec. 28, 1927.

We have seen at times, in severe meningeal conditions, complete absence of involvement of the pacchionian granules as the result of enormous swelling of the cells of the stomas. The fact that many statistics have shown bleeding into the arachnoid space at the time of birth in so many cases may alter our conception of some of the conditions occurring later in life, the most important of which is epilepsy. This group comprised twenty-eight cases.

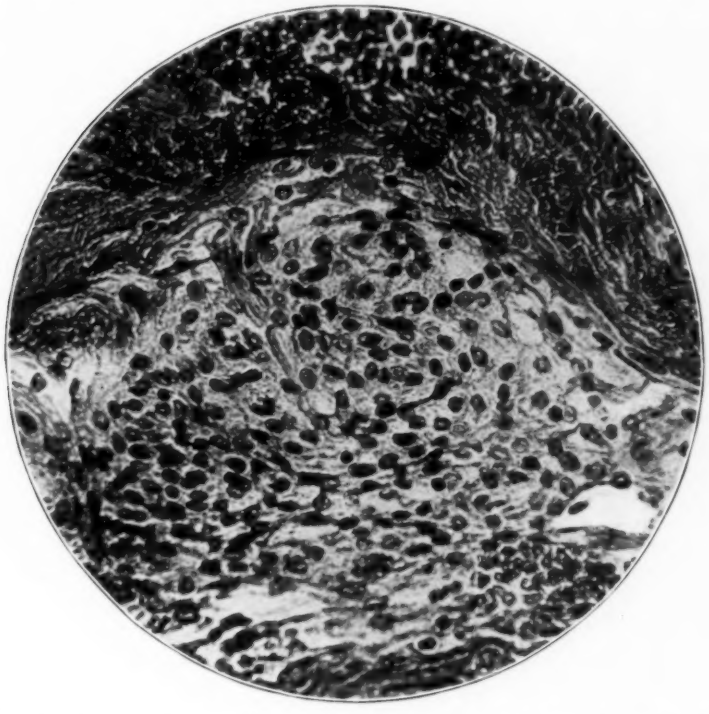

Fig. 9.—Group of mesothelial cells represented in figure 8, under higher magnification.

GROUP 3.—*Hyperplastic Conditions.*—This is the direct opposite of the condition in group 2. There is a great increase in the size of the individual elements. Pathologically, there is an enormous dilatation of the pacchionian granulations (fig. 10) with rounding of their outlines, with enormous central cores of myxomatous material that stain homogeneously with the acid dyes. They contain but few cellular elements, although with the fibrillar stains, as with that of Klarfeld, there is seen a network of fibrils but much more widely separated than is seen in the normal specimen (fig. 11). One can see an enormous number of vacuoles separating the fibrillar network within the pacchionian body,

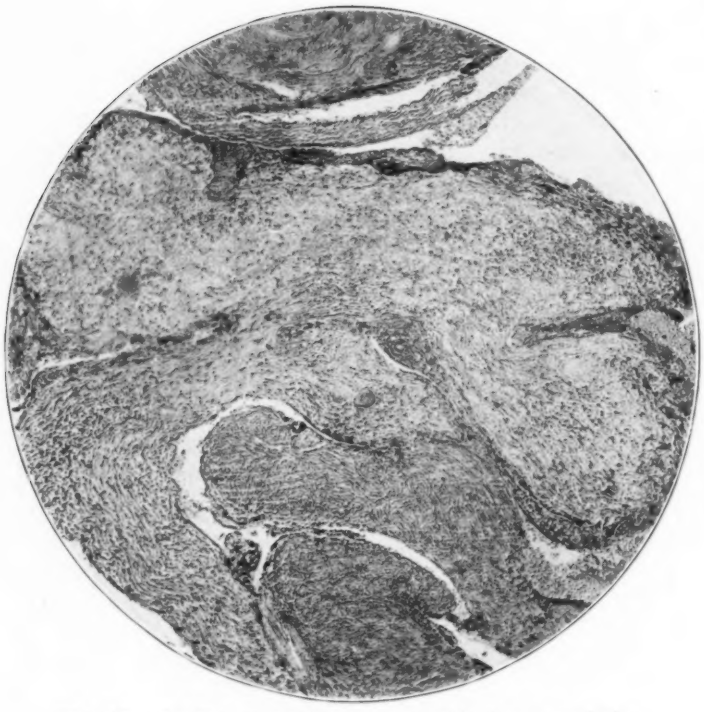

Fig. 10.—Swollen and edematous pacchionian granulations.

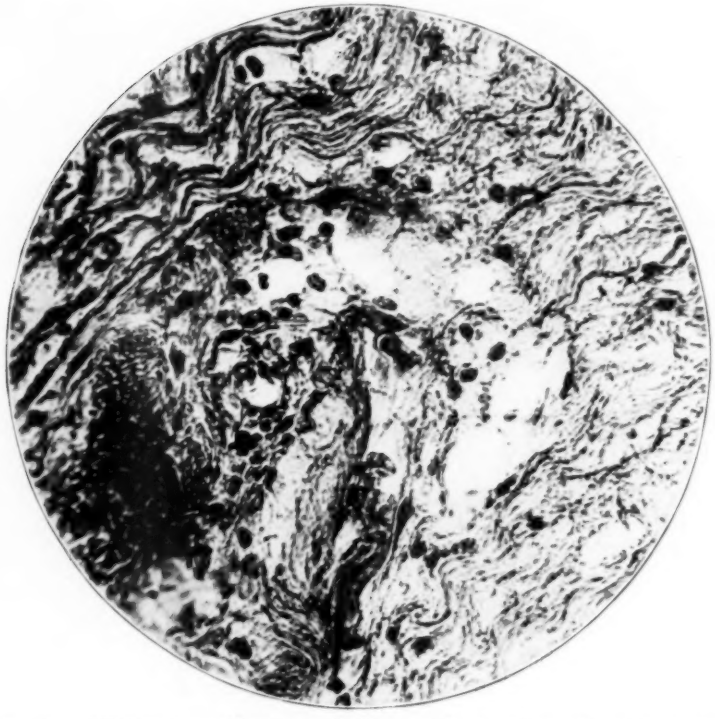

Fig. 11.—Higher magnification of figure 10, showing edematous condition within pacchionian granule.

each containing a finely granular material. The arachnoid covering also shows a wide separation, rarely with clumping of the mesothelial cells as seen under ordinary conditions. At times the swelling is so marked that even the stomas are widely dilated. This condition has been met with characteristically in certain clinical conditions, particularly acute alcoholism and other hydropic conditions of the brain such as acute uremia, in some of which there has been a history of convulsions prior to death. (Six of a total of forty-three cases belonged in this convulsive group.)

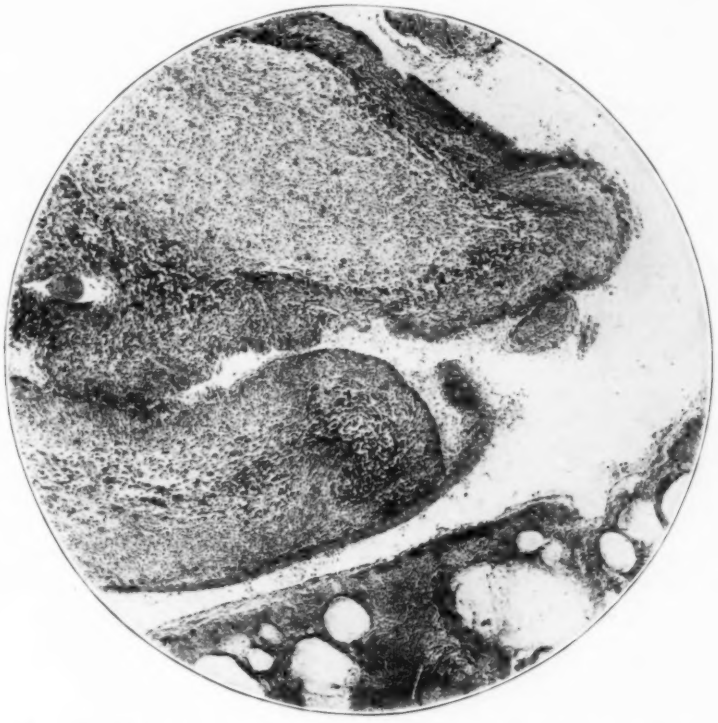

Fig. 12.—Fibrosis and thickening of covering of pacchionian granule.

GROUP 4.—*Fibrosis.*—This consists of forty-two cases. In this group there is not only a fibrosis of varying degree but also an associated atrophy and shrinkage of the pacchionian granulations (fig. 12). So dense may the fibrosis be that the fibrillar stains show a maze of fibrils and make a marked contrast to the edematous type (fig. 13). With high magnification in the fibrillar stains (Klarfeld) one sees clearly the marked fibrillar increase. This fibrillar increase may occur not only within the interior of the pacchionian granule, but may occur only in the covering membranes of the granules. As a rule this condition of fibrosis

of the pacchionian granules has been found in association with fibrosis of the soft membranes of the brain, which, as is well known, occurs in paresis, in the arteriosclerotic conditions of the brain and in chronic alcoholism. The thickening of the arachnoid covering of the granulations is of interest in that Winkelman and Wilson[19] (1925) have demonstrated an increase in the thickness of the arachnoid covering with advancing years.

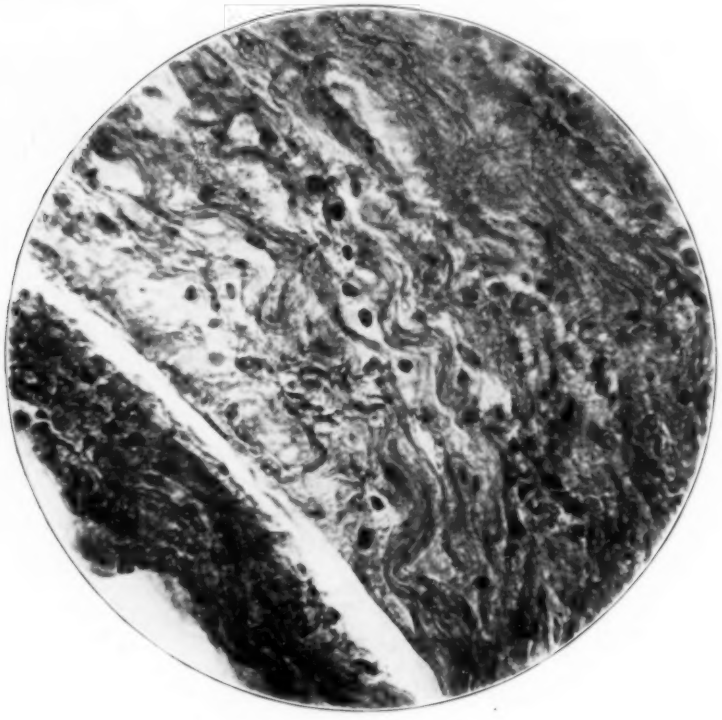

Fig. 13.—Fibrosis within pacchionian granule.

The clinical importance of this change need only be mentioned to be appreciated. It does occur in conditions in which convulsions are of frequent occurrence: paresis, chronic alcoholism and uremia. In these, cortical atrophy is not an unusual observation. In this group a history of convulsions was noted in three cases.

Degenerative conditions can be placed in this group. Figure 14 is a good example of calcification that may occur.

19. Winkelman, N. W., and Wilson, G.: Localized Proliferations of the Arachnoid: Possible Relation to the Origin of Tumors, Arch. Neurol & Psychiat. **14**:46 (July) 1925.

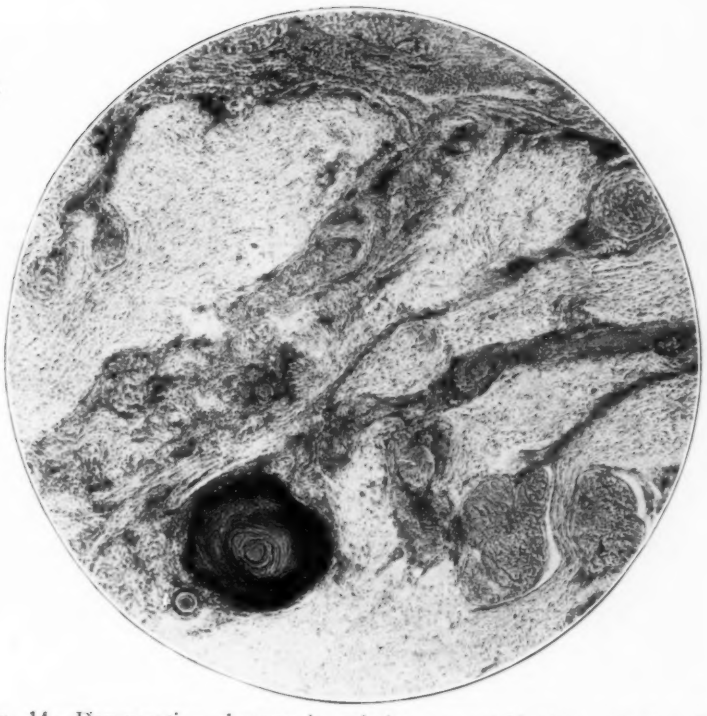

Fig. 14.—Degenerative changes in relation to pacchionian granule. Large concentric calcified body can be seen.

Fig. 15.—Intense distention of pacchionian system by acute inflammatory elements (acute meningitis).

GROUP 5.—*Infiltrative Conditions.*—This group comprises fifty-three cases, in which the same abnormal elements that are to be found in the subarachnoid space are seen in the pacchionian granulations. The most frequent are the inflammatory elements that occur in the meningeal conditions. These include not only the acute forms of meningitis, but the relatively chronic types, such as tuberculosis and syphilis. We have seen the pacchionian granules so packed with inflammatory cells (fig. 15) that they were obviously distended. One can see clearly under higher

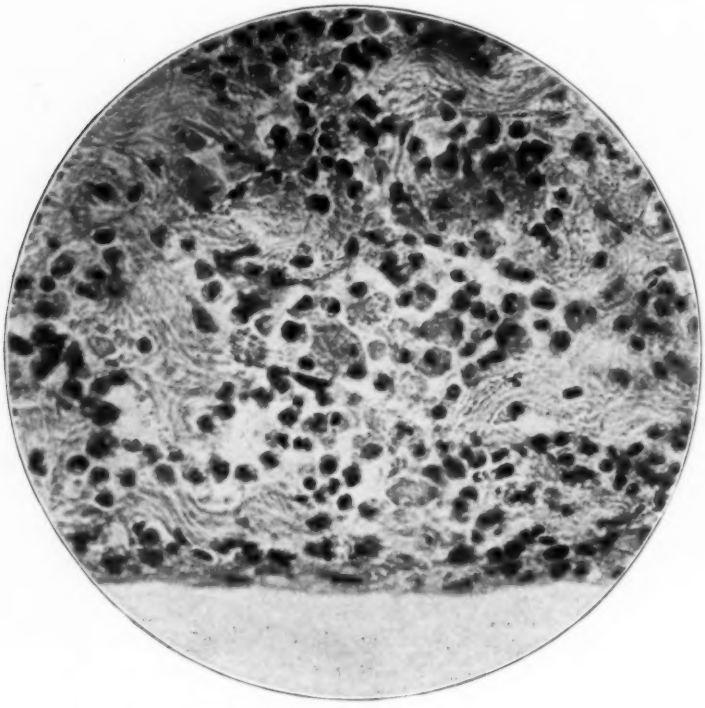

Fig. 16.—Polymorphonuclear cells and macrophages within meshwork of pacchionian granule. Magnification of figure 15.

magnification (fig. 16) that not only are polymorphonuclear cells present, but also large cellular elements (macrophages) containing pigment of various sorts, just as they are to be found in the subarachnoid space in acute meningitis. In syphilis, this inflammatory exudate may be associated with fibrosis as well. It is to be stressed, again, that in many cases there is such swelling of the cells lining the stomas that the inflammatory cells do not enter the pacchionian system.

One of the most interesting of the foreign elements found in the pacchionian granules is blood. We have seen in acute bleeding within

the subarachnoid space, no matter from what cause, the blood elements in the meshes of the pacchionian granulations. Later, blood pigment alone may be seen. We have also seen cases in which the pigment has apparently made its way outside the arachnoid covering, into what is really the subdural space of the individual granulations (fig. 17).

Another foreign element found at times within the pacchionian granule is cancer cells. It is well known that carcinoma can invade the subarachnoid space of the cerebrum in what is known as meningitis

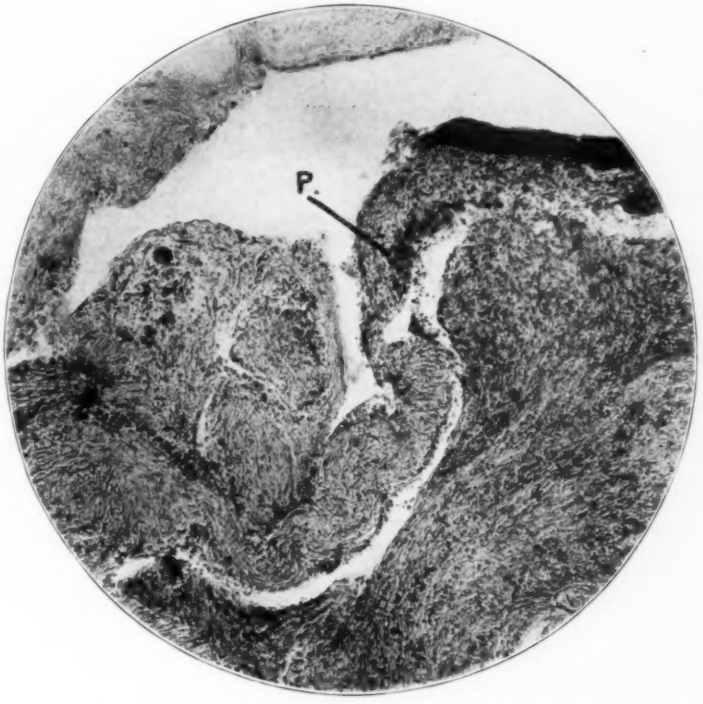

Fig. 17.—Pacchionian body showing blood pigment outside the arachnoid covering, *P*.

carcinomatosa, or what is more properly called meningeal carcinomatosis. There may be no intracerebral nodule formation. While the cancer cells are usually larger than the ordinary inflammatory elements, still some manage to lodge within the pacchionian elements.

COMMENT

The significance of these pathologic observations on the function of the pacchionian bodies must be a subject for further investigation.

It is evident that if the drainage of cerebrospinal fluid is dependent on these structures, and impairment of function can be demonstrated as being due to their pathologic involvement, several obscure clinical

Tabulation of Cases Studied

No. of Cases	Clinical Diagnosis	Pathologic Observations	Convulsions
		Group 1: The Aplasias	
8	Idiopathic epilepsy	Microscopic lesions	Yes
3	Hydrocephalus	Confirmatory	In 1
1	Acute alcoholism	Wet brain	No
1	Cancer meningitis	Cancer of meninges	No
1	Brain tumor	Glioma of cerebrum	No
		Group 2: The Hypoplasias	
	Paresis	Paresis	Occasional
	Angle tumor	Angle tumor	No
	Huntington's chorea	Striatal degeneration	No
	Multiple sclerosis	Multiple sclerosis	No history
	Tuberculosis	Acute meningitis	Yes
	Paralysis agitans	Pallidonigral degeneration	No
	Acute rheumatism	Hemorrhagic "encephalitis"	No
	Tuberculous meningitis	Arteriosclerosis	No
	Alcoholic psychosis	"Central neuritis"	Yes
	Paresis	Paresis	No
	Syphilis	Meningovascular syphilis	No
	Cerebral hemorrhage	Hemorrhage	No
	Cancer of pancreas	Arteriofibrosis	No
	Myelitis	Tuberculous meningomyelitis	No
	Cardiac psychosis	Acute brain changes	No history
	Paresis	Cerebrospinal syphilis	Occasional
	Epilepsy	Severe brain atrophy	Yes
	Epilepsy	Temporal lobe tumor	Yes
	Traumatic epilepsy	Brain atrophy	Yes
	Epilepsy, syphilis	Paresis	Yes
	Dementia praecox	Cortical atrophy	No
	Fracture, skull	Multiple hemorrhages	No
	Dementia praecox	Ganglion cell changes	No
	Brain tumor	Glioma of pons	No
	Manic depression	"Central neuritis"	No
	Pellagra	"Central neuritis"	No
	Hemiplegia	Arteriosclerosis	No
	Senile deterioration	Arteriosclerosis	No
		Group 3: The Hyperplasias	
18	Acute alcoholism	For most part wet brain and acute cell changes	In 2
21	Uremia	Acute cell changes and small vessel changes	In 4
1	Pituitary tumor	Adenoma of pituitary gland	No
1	Veronal poisoning	Severe cell changes	No
1	Parieto-occipital tumor	Glioma	Yes
1	Dementia praecox	Severe edema	No
		Group 4: Fibrosis	
9	Chronic alcoholism	Cortical atrophy; meningeal thickening	In 2
12	Paresis	Paresis	In 1
21	Arteriosclerosis	Arterial changes	None
		Group 5: Infiltrative	
26	Tuberculous meningitis	Tuberculous meningitis	None
18	Acute meningitis	Acute meningitis	None
2	Cancer metastasis	Cancer nodules	None
5	Subarachnoid bleeding	Subarachnoid hemorrhage	In 1
2	Syphilitic meningitis	Meningovascular syphilis	None

manifestations may find a possible explanation. The diffuse collections of fluid existing around the pacchionian bodies in cases associated with evident shrinkage of the brain, and so-called "cortical atrophy" so frequently found at operation, encephalography and necropsy must be considered in the light of a possible pressure atrophy due to this increased

accumulation of fluid which finds insufficient means for escape. The mechanism and pathologic correlation of the "cortical atrophy" noted are considered in detail by us in another paper (1929).

The predisposition to cerebral edema in certain cases of hydration as well as the tendency toward convulsive manifestations in this group has been presented by one of us (Fay,[20] 1928). The part that increased fluid may play in certain types of the convulsive state has led to a clinical experiment based on dehydration which has been carried on for the past two years, with interesting and encouraging results.

We are inclined to view the pathologic changes noted in the subarachnoid villi and pacchionian bodies as fundamentally responsible for many of the slight or extensive gross changes noted, associated with abnormal collections of subarachnoid fluid. The effect of acute and chronic stasis in the circulation of the cerebrospinal fluid directly or indirectly influences intracranial pressure and may thus affect the vascular supply of the brain itself, enclosed as it is within the rigid confines of the skull.

The establishment of a definite physiologic function of the subarachnoid villi and pacchionian granules by Weed has led to our demonstration of a definite pathologic change confined to these structures.

Subsequent investigation and clinical analysis must determine the significance and effect this pathologic change may have on intracranial pressure, and the cerebral and vascular structures affected.

CONCLUSIONS

1. Definite pathologic involvement of the subarachnoid villi and pacchionian granules has been noted in our study of more than 200 human specimens.

2. We have chosen to divide the pathologic changes so far observed into five groups: (*a*) aplastic, (*b*) hypoplastic, (*c*) hyperplastic, (*d*) fibrotic and (*e*) infiltrative.

3. The actual pathologic changes thus demonstrated in these structures may greatly influence the physiologic function attributed to them, and form a basis for the consideration of certain disturbances of the circulation of the cerebrospinal fluid, heretofore obscure.

4. The pacchionian granules, as a result of their direct communication with the subarachnoid space, are subject to the same pathologic changes which occur in cerebrospinal fluid space in general.

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20. Fay, Temple: Some Factors in the "Mechanical Theory of Epilepsy," With Especial Reference to the Influence of Fluid, and its Control, in the Treatment of Certain Cases, Am. J. Psychiat. **8**:783 (March) 1929.

ABSTRACT OF DISCUSSION

Dr. W. G. Spiller, Philadelphia: Dr. Fay has devoted much attention to the study of increased pressure of the cerebrospinal fluid, particularly in conditions of the convulsive states and has obtained observations that have confirmed many of his views. On the pathology of the pacchionian system depends in large measure the correctness of the views that he has entertained.

Dr. Winkelman has stated in his paper, which the time limit did not permit him to read, that Förster refers to the fact that when there is obstruction in the pacchionian bodies the air is not eliminated within the usual period of time in making encephalograms.

I have seen the sections that Dr. Winkelman has prepared and they appear to me to be of great value.

He has pointed out in his paper also that the pacchionian bodies develop at the age of puberty. Many of us probably have seen patients in whom some injury of the head occurred in the first or second year of life, at which time there were no convulsions, but they developed at the period of puberty. His statement that the pacchionian bodies develop at the period of puberty offers an explanation for these cases. When there has been an injury to the head early in life and hemorrhage has occurred, the blood has passed into the pacchionian bodies. Swelling of the cells lining the stomas with degenerative changes have occurred, and in some of these cases the pacchionian bodies have not developed. This explains why so frequently epilepsy first develops at the period of puberty.

It is interesting that in a large number of the authors' cases of so-called idiopathic epilepsy and of hydrocephalus, imperfect or no pacchionian bodies have been found. This can hardly be ignored as an important observation.

This observation also explains why cortical atrophy occurs frequently in cases of hydrocephalus from increased pressure of the cerebrospinal fluid, and this atrophy may be recognized by an encephalogram.

All this work is in confirmation of Weed's work on the physiology of the cerebrospinal fluid. I know from Dr. Winkelman that he has spent a year and a half in the preparation of this work, and that he began it without a study of the literature in order that his mind might not be influenced by preconceived ideas. It was after he had obtained his pathologic observations that he studied the literature carefully.

I do not believe that either Dr. Fay or Dr. Winkelman would assert that they had solved all the problems of the epilepsies, but I think that they have made an important contribution to the subject.

VILLI (PACCHIONIAN BODIES) OF THE SPINAL ARACHNOID*

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Arachnoid villus is the name given by Luschka [1] to a protrusion of the arachnoid membrane and the underlying spongy subarachnoid tissue. A hypertrophied villus consisting of several lobules or villi covered by a common membrane and possessing a common stalk is known as a pacchionian body. Both the villi and the pacchionian bodies or granulations are of common occurrence in the cerebral arachnoid around the meningeal veins and dural sinuses, especially the superior longitudinal. Both consist of loose areolar connective tissue which is dense near their summits and extends to the stalk or pedicle of the villus and from here to the subarachnoid space (fig. 1).

The fibers of the connective tissue are covered by mesothelial cells which also cover the summits of the villi, extending to and being continuous with the mesothelial cover of the rest of the arachnoid membrane. Within the villi these cells often form clusters, but they are often scattered singly. The villi are devoid of vessels; they usually invade the dura, where they are lodged within lacunae—round or oval spaces in the dural interstices. They also invade the dural venous sinuses, where they float, as Key and Retzius [2] put it, or, in their hypertrophied form as pacchionian bodies they may pierce the dura and invade the bony tissue itself. According to Trolard,[3] villi may be present without lacunae, and furthermore it is not unusual to find in some lacunae, instead of connective tissue villous growths, clusters of proliferated endothelial or rather mesothelial cells. In other words, the interstices of the dura may be separated not by villi, but

* Submitted for publication, May 1, 1929.

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* Read at the Fifty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 28, 1929.

1. Luschka: Ueber das Wesen der Pacchionischen Drüsen, Müller's Arch. f. Anat. u. Physiol., 1852, p. 101.

2. Key, A., and Retzius, G.: Studien in der Anatomie des Nervensystems und des Bindegewebes, Stockholm, P. A. Norstedt & Söner, 1876, vol. 1, p. 179.

3. Trolard: Les granulations de Pacchioni. Les lacunes veineuses de la dure-mère, J. de l'anat. et de la physiol. **28**:28, 1892.

by their equivalents. Schmidt [4] called them "epithelial tufts." These originate from the mesothelial cover of the arachnoid and are looked on as forerunners of the pacchionian bodies, as their embryonic form. As such they are present even in the new-born infant, and they are supposed to be the principal source of meningeal tumors (endotheliomas or meningiomas).

Since the days of Key and Retzius, the arachnoid villi were generally considered the main avenues of escape or absorption of the cerebrospinal fluid—but only from the cerebral subarachnoid cavity.

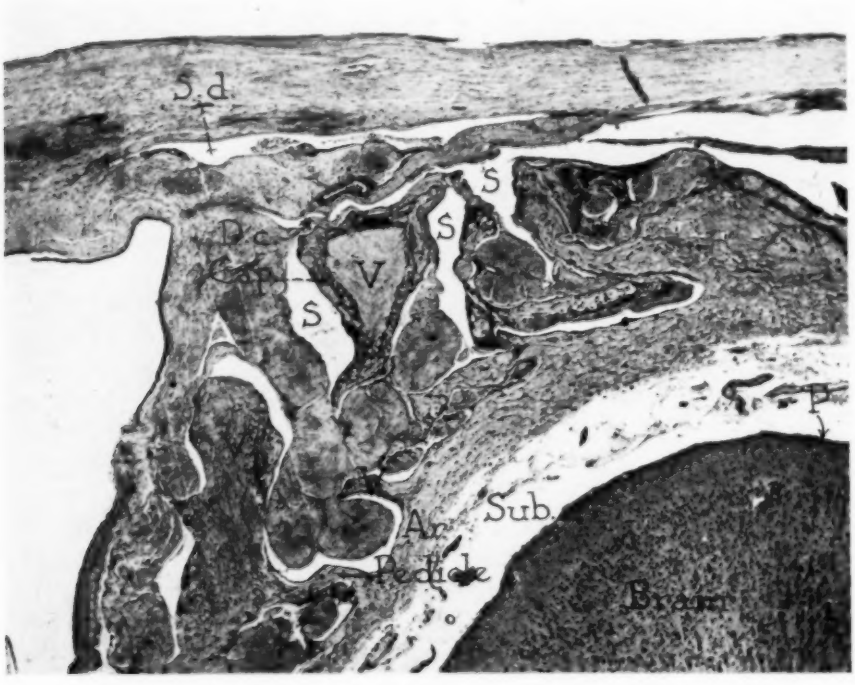

Fig. 1.—Brain. *S. d.* indicates the subdural space; the dura is above in the form of a broad band sending prolongations among which spaces (*S, S, S*) are seen; these are probably the so-called venous lacunae harboring villi (*V, V, V*). One to the left shows an abundance of mesothelial cells; the one in the middle shows a capsule (*Cap*) made up of mesothelial cells; this capsule is covered by a dural capsule (*D. c.*); *Ar.*, arachnoid membrane; *P*, pia, and *Sub.*, the subarachnoid space. Van Gieson stain; × 33.

The mode of escape of fluid from the spinal subarachnoid space was believed to be through different channels, mainly through the perineural spaces of the spinal roots. Such different pathways had to be

4. Schmidt, M. D.: Ueber die Pacchionischen Granulation und ihr Verhältniss zu den Sarcomen und Psammomen der Dura Mater, Virchows Arch. f. path. Anat. **170**:420, 1902.

considered, for it has generally been believed that neither pacchionian bodies nor villi are present in the spinal arachnoid. When they were mentioned (Odyniec,[5] Bluntschli[6]) in the spinal membranes, it was rather in the form of an allusion to the fact that the mesothelial cells of the spinal arachnoid may form clusters and nodules which bear a resemblance to the arachnoid nests of the cerebral dura. As the latter are considered embryonic villi (Schmidt[4]), a conclusion was permissible that similar formations in the spinal arachnoid are an early, undeveloped stage of a villus. Elman[7] is much more precise and clear.

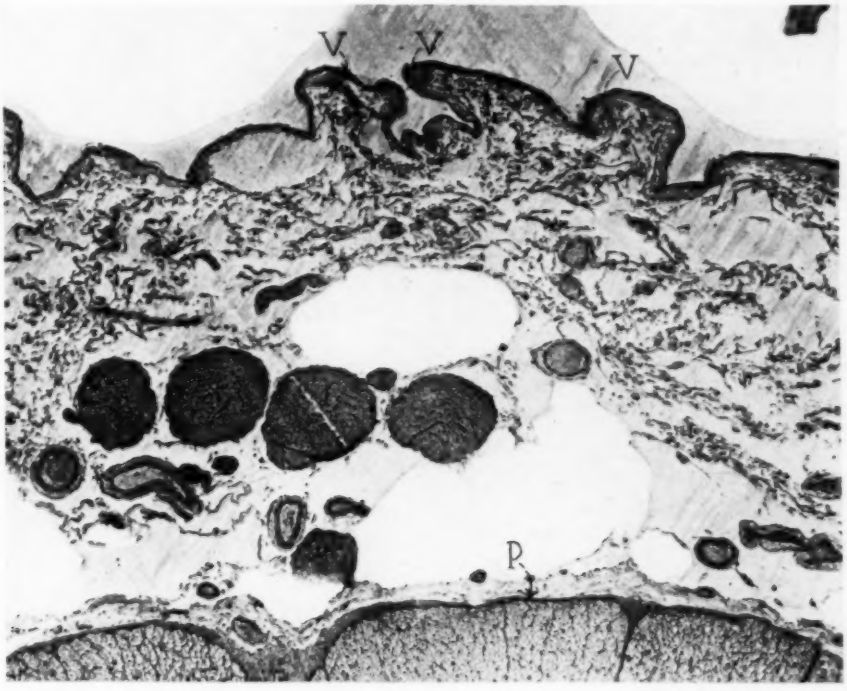

Fig. 2.—Spinal cord. *V*, *V*, *V* represents the villi or projections of the arachnoid membrane; *P*, marginal glia of the spinal cord; the band above is the pia; between the pia and the *V*, *V*, *V* is the subarachnoid space containing numerous fibers which are invading the villi. The round bodies are the cross-sectioned posterior roots. Toluidine blue stain; × 33.

5. Odyniec, A.: Die Endothelknoten in der Arachnoidea Spinalis und ihre pathologische Bedeutung und die Genese der Sandkörperchen, Inaug.-Diss., Zurich, Leeman & Company, 1908.

6. Bluntschli, H.: Versuch einer Phylogenese der Granulationes Arachnoidales (Pacchioni) bei den Primaten, Verhandl. d. Gesellsch. deutsch. Naturf. u. Aerzte **80**:363, 1908.

7. Elman, R.: Spinal Arachnoid Granulations with Especial Reference to the Cerebrospinal Fluid, Bull. Johns Hopkins Hosp. **34**:99 (March) 1925.

He pointed out that in dogs and guinea-pigs clusters or columns of mesothelial cells are present at the point of reflection of the arachnoid membrane over the emerging spinal roots, where the arachnoid forms an angle. Here the clusters of the cells are blended with the contiguous dural prolongations, and in many instances actually invade the fibrous substance of the dura. According to Elman, they are

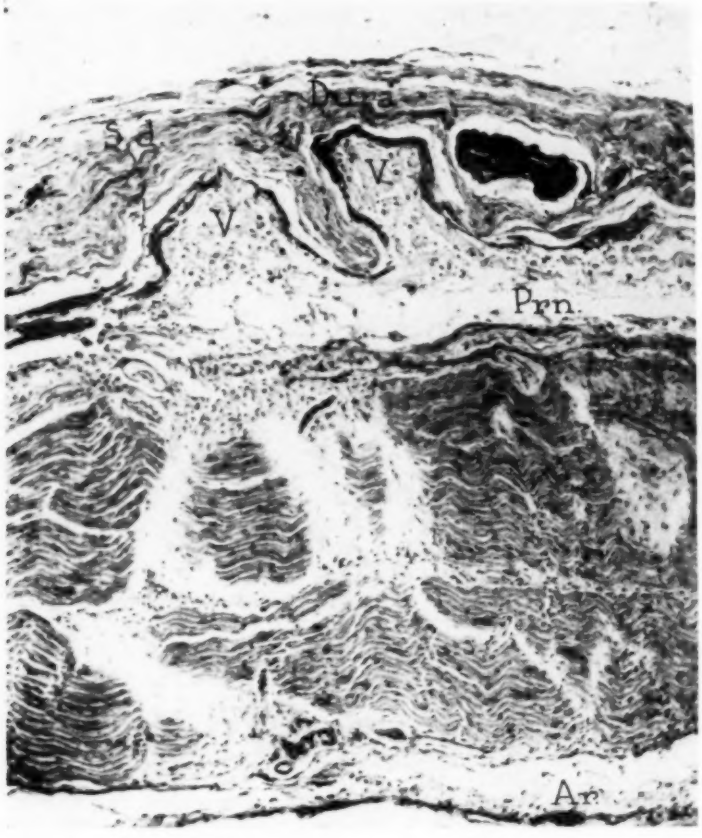

Fig. 3.—Spinal cord. The dura is invaded by villi (*V, V*); *S. d.*, subdural space; *Prn.*, subarachnoid or perineural space of the posterior root—bundles of longitudinal nerve fibers; *Ar.*, arachnoid; the dark body within the empty space to the right is a section of the villus (its dark summit). Hematoxylin and eosin stain; × 80.

analogous to the arachnoid granulations of the cerebrum and "permit the passage of foreign solution, while the remainder of the arachnoid membrane, under the same circumstances, remains impermeable." In his injection experiments, Elman could not demonstrate the existence of "fluid pathways physiologically connecting the subarachnoid cavity with

the so-called perineural spaces." Like Sicard and Cestan,[8] he considered the latter closed.

The foregoing data are in flat contradiction to the classic studies of Key and Retzius. These investigators held that the mode of escape of the fluid from the spinal subarachnoid is by way of the perineural root spaces; from the cerebral cavity it is by way of the pacchionian bodies, and also, but partly, by way of the perineural spaces of the cerebral nerves.

The discrepancy between the dominant views concerning the pathways of discharge of the cerebrospinal fluid from the brain as compared with that from the spinal cord is apparent; these views are based largely on physiologic experiments. Pathologic facts or studies, so far

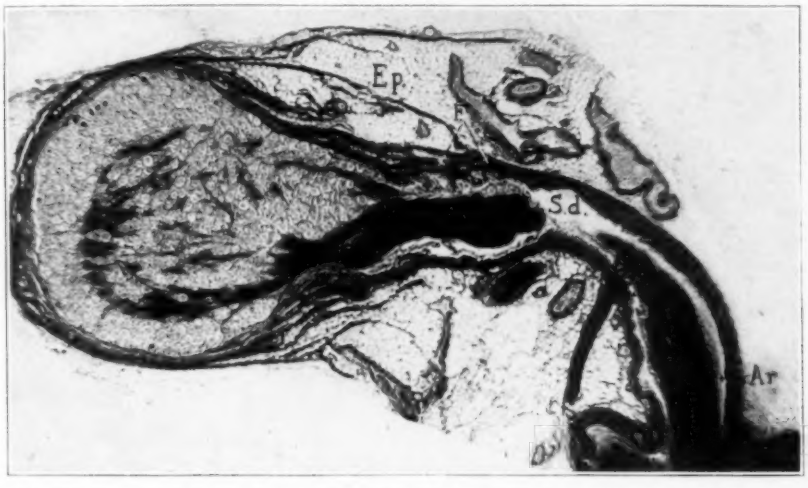

Fig. 4.—*Ar.* represents the arachnoid accompanying the root fibers with which it enters the spinal ganglion; *S. d.*, subdural space; *F*, folds of the dura which are continuous over the ganglion; *Ep.*, epidural space. Weigert and van Gieson stain.

as I am aware, have been wholly neglected. *A priori,* it would seem plausible that the structure of the cerebral and spinal arachnoid membranes should be alike and that the mode of escape of the cerebrospinal fluid should be similar. As a matter of fact, the discrepancy between the structure of the cerebral and that of the spinal dura-arachnoid is only apparent. Over the convexity of the brain these membranes, especially the dura, appear rather smooth, over the spinal cord they do not. Extending over a long stretch of the latter, both the arachnoid

8. Sicard, J. A., and Cestan, R.: Étude de la traversée meningo-radiculaire au niveau du trou de conjugaison. Le nerf de conjugaison, Bull. et mém. Soc. méd. d. hôp. de Paris **21:** (3d series) 715, 1904.

and the dura must of necessity make frequent reflections over the spinal roots as well as the ganglia. In the brain such conditions exist only at the base, around the gasserian ganglion and the cranial nerves.

In figure 1 the arachnoid villi (*V, V, V*) of the brain are depicted in their relationship to the dura, its septums and sinuses, and the subarachnoid space. At *D.c.* is the dural capsule dividing the villus from the so-called dural venous sinus (also called by some venous lacunae). The numerous mesothelial cells (*Cap*) investing the summits of the villi are well shown underneath. The spinal fluid, according to

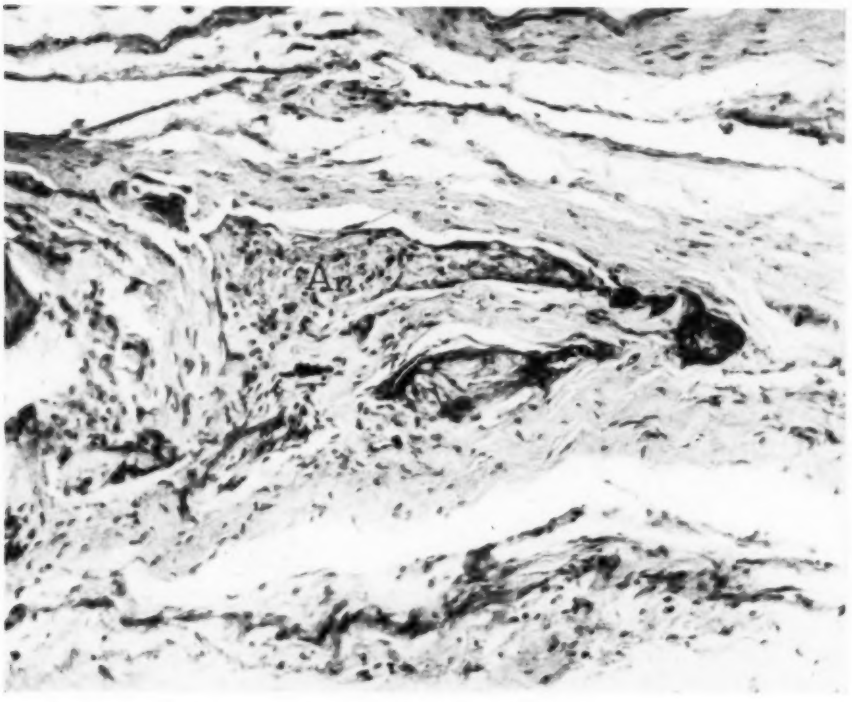

Fig. 5.—*Ar.* indicates nests of mesothelial cells enclosed within the dura. Toluidine blue stain; × 150.

popular conception, travels from the subarachnoid space (*Sub.*) to the villus, through its pedicle, and enters the "venous sinuses" of the dura (*S, S, S*) through the thin mesothelial cover and the dural cap. From here it is removed to the general blood circulation. In figure 2, the spinal villi are shown in the form of projections (*V, V, V*) from the arachnoid. They strongly resemble the cerebral villi not only in appearance, but also in structure. Like the former, they consist of bands of connective tissue fibers among which various cells, especially mesothelial, are scattered; the summits of the villi are stained darker than their bulk.

This is especially well shown in figure 3. Here not only the protrusions (*V*, *V*) are seen, but also their actual invasion of the dura, thus resembling the condition presented in figure 1. The pedicle here is somewhat broader and the connective tissue covered by the mesothelial cells extends from here to the villus as well as to the subarachnoid perineural space (*Prn.*). It is divided from the dura by a free sub-

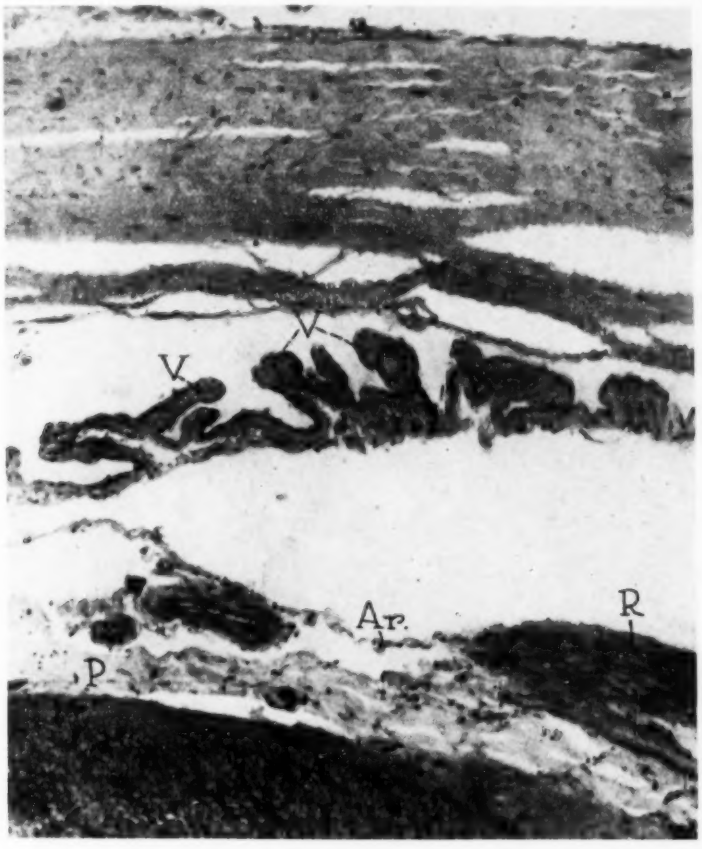

Fig. 6.—*V*, *V* represents the villi artificially separated from the arachnoid (*Ar.*); *R*, posterior root; *P*, pia covering the spinal cord. Toluidine blue stain; × 110.

dural space (*S.d.*) which can be followed up to and around the spinal ganglion (fig. 4). Here, as figure 4 shows, the root fibers (dark bundles in the photograph) accompanied by the arachnoid (*Ar.*) enter the spinal ganglion which is invested only by the dura. No "reflection" of the arachnoid over the posterior root, as described by Elman in dogs, is seen here. The irregular margin at *F* is a fold of the dura

which before reaching the spinal ganglion is divided from the subjacent arachnoid by the subdural space (*S.d.*). The latter appears over the spinal ganglion as a mere, but clear, tissue space. In photomicrograph 3 the cell elements within the villus are especially well seen. Under a higher power lens some of them showed a pale rim of cytoplasm. Other nuclei were within bipolar cells (fibroblasts); much more numerous were naked nuclei, oval or round, without cytoplasm, scattered singly, or forming clusters (mesothelial cells). They were mixed with numerous small, round, homogeneous and pale fragments

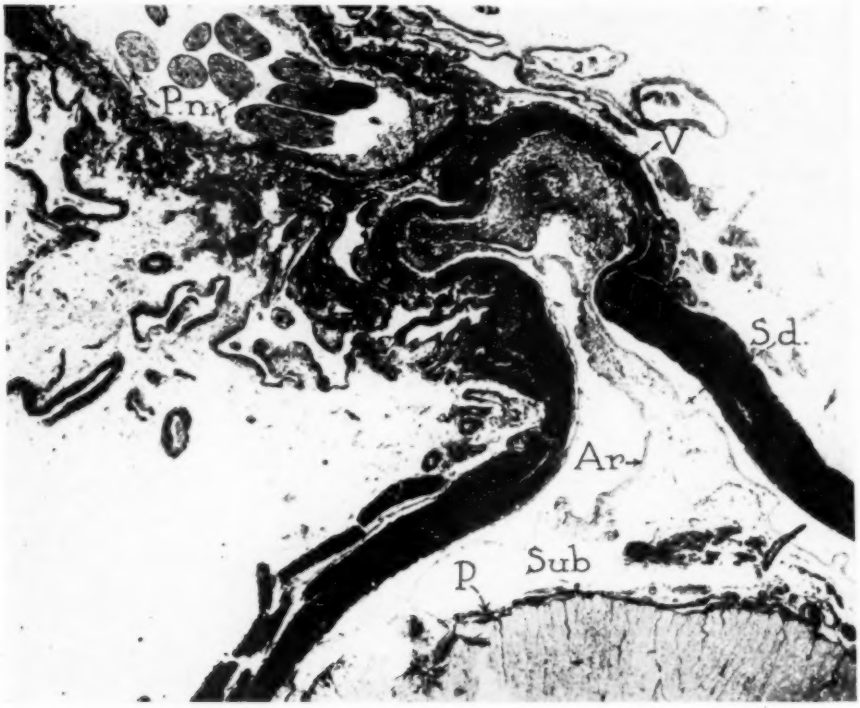

Fig. 7.—The dark band is the dura; *S. d.*, subdural space obliterated above by the hyperplastic villus; *Ar*, arachnoid membrane; *Sub*, subarachnoid space; *P*, pia of the spinal cord; *P. n.*, peripheral nerve crossing the epidural space. Bielschowsky counterstained with Alzheimer-Mann stain; × 20.

of the connective tissue fibers which made up the bulk of the arachnoid villus. Blood vessels were absent here, as in the cerebral villus. Near the summit of the villus the foregoing cell bodies (mesothelial cells, fibroblasts and polyblasts) were much more numerous and denser, and often formed rather thick cell layers. Psammoma bodies (corpora arynacea) were occasionally present.

As stated, the surface of the villus is divided from the superimposed dura by a free space. In many instances, however, the villus is blended with the dura. In figure 5, for instance, masses of mesothelial cells (*Ar.*) are enclosed within the (spinal) dura, much resembling the arachnoid nests of the cerebral dura that have been mentioned previously. In the spinal dura such nests, however, are less common and less typical; furthermore, they are not enclosed within lacunae which are

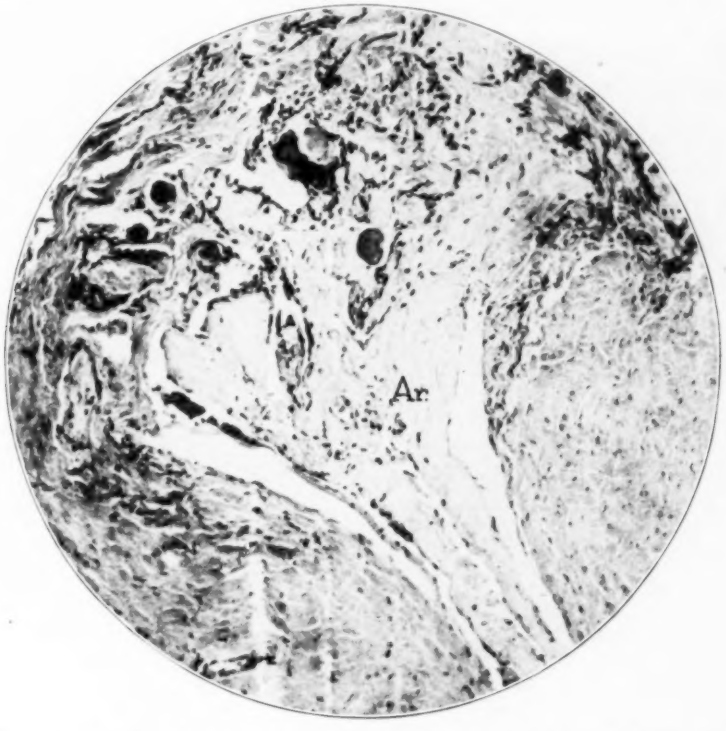

Fig. 8.—Tabes. The villus of the arachnoid (*Ar.*) membrane is hypertrophied, fibrous and appears as a pacchionian body which above is blended with the dura; the stalk of the body is separated to the right and left by the subdural space; several psammoma bodies are present in the upper portion of the hypertrophied villus. Van Gieson stain; × 110.

so abundant in the cerebral dura. Venous sinuses, which are so characteristic for the cerebral dura, are lacking in the spinal dura.

The shape, size and number of the spinal villi vary greatly. They may be numerous (fig. 6) and often appear as mere threads or folds separated from one another by regular intervals. In other instances, again, they may be immensely rich in connective tissue, appear hyperplastic (fig. 7), fibrous (fig. 8), adherent to the dura and as obliterating

the subdural space (fig. 9). In such cases they appear as veritable pacchionian bodies, well shown in figure 8 from a case of tabes. In figures 8 and 9 the summit of the hypertrophied villus is branched and covered by whorls of mesothelial cells invading and obstructing the subdural space above, while in figure 7 the folded dura mater merely harbors a folded arachnoid but is not invaded by it. Both folded membranes run parallel, separated from one another by the prolongations of the subdural and subarachnoid spaces. In such cases one can speak of hypertrophied villi, as the intruded folded arachnoid appears

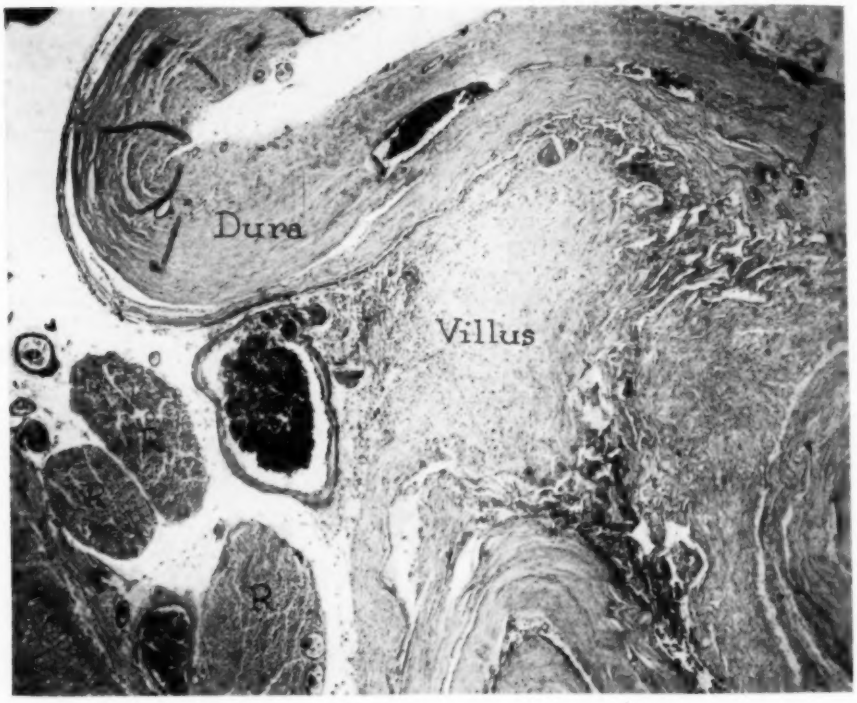

Fig. 9.—Between the dura and the underlying villus are whorls of arachnoid with mesothelial cells. *R, R, R* are the spinal roots with the pia and spinal cord underneath.

hyperplastic and is much richer in connective tissue than the adjacent subarachnoid areas. They often occur in the brain, especially in older people, and are commonly known as pacchionian bodies.

COMMENT

Contrasting the villi pictured in figures 2 (senility), 3 (multiple sclerosis), 7 and 8 (tabes dorsalis) and 9 (cerebellar cyst), one can see differences in them. In multiple sclerosis they are especially note-

worthy. Here they are smaller and rather slender, somewhat rich in cells and do not invade the subdural space above as do the villi pictured in figures 8 or 9. The differences in size, cell and tissue contents, and relationship to the subdural space may be due to the effect exercised on them by the cerebrospinal fluid, which in tabes is different from that in multiple sclerosis. The cerebrospinal fluid enters the villi from the subarachnoid space of the spinal roots (perineural root spaces), which are well shown in figure 3 (*Prn.*). From over the roots the space can be followed along their entire length to the spinal ganglion, as can be seen in figure 4.

These observations are in flat contradiction to the conclusions drawn by Elman from his experiments. Making injections of india ink, but mainly iron ammonium citrate and potassium ferrocyanide, into guinea-pigs and dogs, according to Weed's technic, Elman came to the conclusions that: the perineural spaces pictured are not constant; they were absent in cords that had been dehydrated quickly in twenty-four hours; the arachnoid membrane in a dog is continuous "over a very short distance, one millimeter or two, then becomes reflected back on the proximal side of the nerve root to become continuous with the pia mater." The subarachnoid space, he stated, forms a closed cavity at this point and is not connected with the nerve roots. The contents of the subarachnoid space cannot, for this reason, be discharged to the periphery along the perineural pathways and the peripheral nerves as had been maintained by Key and Retzius.

Long before Elman, Sicard and Cestan[8] arrived at the same conclusion. Though they used considerable force in injecting india ink into the subarachnoid space, they could not force it through beyond a certain distance in the perineural root space. Yet it is interesting to note that the excessive force of the injections was blamed by Elman for the successful injections of Key and Retzius.

The observations here outlined are in complete accord with those of Key and Retzius, for the photomicrographs show that the perineural space is continuous with the spinal subarachnoid cavity, that the latter is not a closed space and that the spinal fluid is discharged from the spinal subarachnoid to the perineural space. The observations of Key and Retzius were only partly confirmed by Weed's experiments.[9] With injections of ferrocyanide solutions Weed found "an obvious perineural deposit of precipitated granules which can be followed a short distance outward along the anterior and posterior roots." Yet none of these

9. Weed, L. H.: Studies on Cerebrospinal Fluid, J. M. Research **31**:21, 1914; Article 3, p. 90.

investigators nor other students of this problem (Trolard,[3] Cushing,[10] LeGros Clark,[11] Gladstone and Dunlop[12]) described the villi in the spinal arachnoid; in fact, they denied their existence.

Though considered essential in the brain to enable the cerebrospinal fluid to escape, the villi were evidently considered not necessary in the spinal cord. Here a different mode of escape of the fluid was thought to be at play. The question arises whether this different pathway, through the perineural spaces, is not instrumental in the escape of the fluid also from the brain, and whether the villi or the pacchionian bodies are necessary or essential factors in the process of absorption of the cerebrospinal fluid. A number of facts seem to favor a view that neither the brain nor the spinal cord utilizes the foregoing structures for the purposes of elimination of the cerebrospinal fluid. For instance, the arachnoid villi and their hypertrophied form, the pacchionian bodies, being mere folds of the arachnoid membrane, must possess the same function as the latter. Therefore, if the villi were the organs of absorption of the spinal fluid, this would be also the function of the entire arachnoid—a function nobody would even suggest. As a matter of fact, the villi have no function. As folds they are merely of some significance, just as folds or wrinkles of the skin are of significance, indicating probably an advanced age. Being a soft, thin, delicate membrane made up of loose connective tissue, the arachnoid is more amenable to changes of form than the unyielding dura or pia. Such changes showing as protrusions, thickenings and similar conditions may, as hinted long ago by Faivre (according to Trolard[3]), be brought on by pressure of the subarachnoid contents—the cerebrospinal fluid. When the pressure is excessive, it may affect the shape of the arachnoid, causing the villous protrusions. As near the dural sinuses the pressed on arachnoid meets with little resistance, the arachnoid protrusions (villi) become particularly prominent, forming veritable culdesacs. The spinal fluid enclosed within the latter may be forced through their walls by the continuous fluid pressure in the subarachnoid space (pressure by vis a tergo) and discharged in the venous sinuses. In the spinal cord, the dura possesses a wide epidural space. This favors formation of folds into which the arachnoid follows (fig. 7) as if pushed in by the underlying spinal fluid. As figure 7 shows, it differs in no way from a typical hypertrophied villus of the brain.

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10. Cushing, H.: Studies on the Cerebrospinal Fluid, J. M. Research **31**:1, 1914.

11. LeGros Clark, W. E.: On the Pacchionian Bodies, J. Anat. **55**:40, 1921.

12. Gladstone, R. J., and Dunlop, H. A.: A Case of Hydrocephalus in an Infant, with Comments on the Secretion, Circulation and Absorption of the Cerebrospinal Fluid, J. Anat. **61**:360 (April) 1927.

Because of the peculiar anatomic relationship of the villi to the so-called venous spaces—lacunae and dural sinuses—it was thought that the contents of the villi are discharged into these spaces. Lodged within the lacunae (spaces in the dural wall) the villi project into the sinuses, which are wide spaces between the folded dural membranes. In the dural sinuses the contents of the villi (cerebrospinal fluid) are divided from the blood by a thin membrane. Through this it was supposed that the cerebrospinal fluid escapes into the blood by osmosis, or rather filtration (Weed). As in the spinal dura neither lacunae nor dural sinuses exist, one may assume that the foregoing arrangements in the brain—lacunae, villi, venous dural sinuses—are not for the purpose of absorption of the cerebrospinal fluid. Around the sinuses the dura happens to be full of meshes and is here unopposed or less opposed to the growth of the arachnoid folds than is the unyielding dura at points remote from the sinuses. The fact that the villi often, especially when hypertrophied, pierce the dura, invading the bony tissue itself, would be inconsistent with the theory of the discharge of the fluid into the sinuses, which do not exist above the dura. For the foregoing reasons the villi, though present in both the cerebral and the spinal arachnoid, can hardly possess an absorptive function (by filtration or osmosis). The perineural pathways of the roots in the spinal cord and of the basilar nerves in the brain are evidently of paramount importance. It is probable that they are the main, if not the sole, modes of escape of the cerebrospinal fluid from both the brain and the spinal cord. This problem is being studied more extensively and will form the subject of a special communication.

SUMMARY AND CONCLUSIONS

The spinal arachnoid possesses villi that are analogous to those of the cerebral arachnoid; when hypertrophied, these are veritable pacchionian bodies. Like the cerebral villi, the spinal villi harbor cerebrospinal fluid. They are numerous over the roots, where they are continuous with the perineural root spaces. While filtration or osmosis may possibly take place from the villi into the venous dural sinuses, the main mode of escape of the cerebrospinal fluid is probably by way of the perineural root or nerve spaces. Like the subdural, the subarachnoid cavity is not a closed but an open space.

In conclusion, I wish to emphasize the importance of the study of the meninges. Both spinal cord and brain sections should include all the three membranes, the nerve roots and the ganglia. When this is impossible, the latter should be sectioned separately. Sections made in this manner can alone enable one to follow the channels connecting the spinal cord with the perineural spaces. This will lead to a better

knowledge of the circulation and absorption of the spinal fluid and their bearing on clinical phenomena.

ABSTRACT OF DISCUSSION

DR. N. W. WINKELMAN, Philadelphia: Dr. Hassin is to be congratulated on this work. It is interesting. He deserves considerable credit for advancing a new view even though there may be difference of opinion as to its interpretation. The view that Weed has advanced as to the function of the pacchionian bodies has been practically accepted. I do not think that the work Dr. Hassin has done would altogether negative it, because one sees in any increase of intracranial pressure small bodies projecting through the dura of the brain without relation to sinuses, as shown so well by Cushing. It fits in also with the work that he has done on tabes dorsalis.

TUMORS OF THE CAUDA EQUINA

THE DIFFERENTIAL DIAGNOSIS BETWEEN NEW GROWTHS AND INFLAMMATORY LESIONS OF THE CAUDAL ROOTS *

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This study of the clinical features of lesions of the cauda equina is based on an analysis of forty-five cases from the Surgical Division of the New York Neurological Institute. Twenty-eight of the patients had new growths which involved the roots of the cauda equina; in the remaining seventeen, no tumor was found and the cases were mostly recorded as inflammatory lesions. Two cases of extensively enlarged veins between the caudal roots, which were more or less completely excised, were included in the tumor group because the enlarged blood vessels encroached on the roots and acted mechanically as if they were new growths.

In the seventeen patients with inflammatory changes of some or all of the caudal roots, exploratory laminectomy was performed either because a preoperative diagnosis of tumor had been made or because a new growth could not be excluded. In these patients, the roots were found to be swollen and altered in color, so that an inflammatory process seemed most probable (fig. 1). In some instances, the surgical intervention followed soon after an injection of iodized oil (40 per cent), and the inflamed and swollen appearance of the roots of the cauda equina may well have been due to the irritant effects of that substance. The diagnosis "primary" cauda equina neuritis is not always firmly founded, for nerve roots may be congested and swollen secondary to a tumor or may be only a part of an inflammatory process either inside or outside of the dura.[1]

With the reservations just made, the cases of this series have been divided into two groups—tumors and neuritis—and the clinical features of the two contrasted.

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* Submitted for publication, May 1, 1929.

* Read at the Fifty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 28, 1929.

* From the Surgical Division of the New York Neurological Institute.

1. This was demonstrated by one of our cases in which an exploration of the cauda equina revealed only swollen and congested nerve roots. The patient died several years later from a generalized sarcomatosis with a large extradural tumor in the lumbosacral region.

Synopses of the typical clinical records of two patients with tumors and two with neuritis are given in the following case reports.

REPORTS OF CASES

CASE 1.—*Symptoms of nine years' duration; laminectomy and removal of large tumor of cauda equina. Recovery.*

History.—C. B., a girl, aged 17, was admitted on Feb. 26, 1928. The onset of the symptoms dated back to a fall on the back nine years before, after which she began to suffer from attacks of pain in the lower part of the back which radiated down the left and also the right lower extremity. The pain was felt

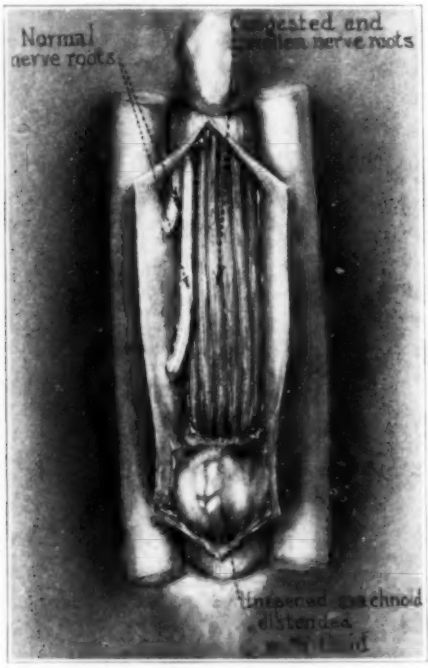

Fig. 1.—Neuritis of the cauda equina with adherent arachnoid (elaborated from surgeon's sketch).

almost daily, would last for several hours, was sometimes very severe, and was made worse by movement or on coughing or sneezing. After the attacks of pain had occurred for four years, a beginning loss of power in the lower limbs was observed. While at play, the child would sometimes fall to the ground on account of sudden loss of power in the lower limbs, but after a short rest the strength of the limbs would return. She became gradually worse, however, and by the end of 1927, the pain was practically continuous. After a lumbar puncture done at that time, she lost almost all power in the lower extremities and began to have urinary difficulties.

Physical Examination.—The patient was poorly nourished and bedridden. She could not raise the left lower extremity but could lift the right a few inches from

the bed. The thighs could not be abducted and adduction was very weak; the power to flex the feet dorsally at the ankles was almost completely lost, but she was still able to move the toes.

Atrophy of the muscles of both lower limbs was more advanced on the left side.

The abdominal reflexes were easily elicited and were not diminished. All of the tendon reflexes in both lower extremities were lost.

Sensory signs were slight and vague. At times the sensation over the sacral dermatomes seemed diminished; at other times, no disturbances of cutaneous sensibility could be found. There seemed to be a slight hyperesthesia at the level of the groins, but this was so variable and indefinite that it was doubtful whether there was any actual sensory disturbance. Vibratory and muscle joint tendon sense were perfectly preserved. Pressure on the left side of the spinous processes

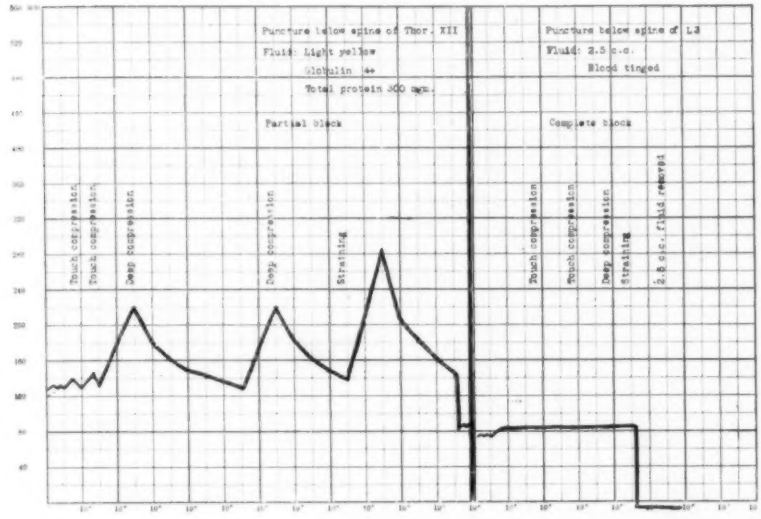

Fig. 2 (case 1).—Tumor of the cauda equina. Manometric chart showing partial block on puncture below the twelfth thoracic spine, and complete block below the third lumbar spine.

of the fourth and fifth lumbar and upper sacral vertebrae caused severe pain which radiated into the left lower extremity.

On lumbar puncture below the spine of the fourth lumbar vertebra, manometric tests showed a complete subarachnoid block, and only 2.5 cc. of blood-tinged fluid could be withdrawn. A second puncture, done below the spine of the twelfth thoracic, revealed a partial block; the fluid was xanthochromic, gave a 4+ globulin reaction, and contained 300 mg. of protein (fig. 2).

Operation and Course.—The arches of the second, third, fourth and fifth lumbar vertebrae were removed, and a large intradural tumor, 5 cm. in length and 2 cm. in width, was removed in fragments from between the roots of the cauda equina. The growth was enclosed in a thin and friable capsule, and was not vascular, so that there was little bleeding during its removal. It had the typical appearance of a "giant" tumor and histologically was a mixed tumor of mesodermal origin—a myxofibroblastoma. The patient made a satisfactory and practically complete recovery. She was discharged on March 30, 1928.

CASE 2.—*Symptoms of five years' duration. Laminectomy and partial removal of tumor of cauda equina. Recovery.*

History.—W. U., a man, aged 32, a printer, whose past history was unimportant except for rather frequent attacks of tonsillitis, an infection of one maxillary antrum and an attack of "lumbago" in 1922, was admitted to the hospital in August, 1928, complaining of pain in the back and legs, associated with a feeling of heaviness in the calves and toes and a burning sensation in the soles. The patient had always worked steadily until August, 1922, when he was bedridden for two weeks on account of pain in the back, which was called lumbago. The pain had been relieved by strapping the back, and had disappeared gradually. He had been completely free from all symptoms until the spring of 1926 when the same pain returned, and from that time on it occurred at varying intervals and was always worse in warm weather. For the four months preceding the patient's admission to the hospital, the pain had been continuous and steadily increasing in severity. It was described as a sharp, shooting pain that would begin in the back and radiate to the front and lower parts of the abdomen and downward into the thighs. At the same time that the pain became continuous, the patient began to experience a sensation of numbness and a "lifeless" feeling in the legs, with burning in the soles of both feet. Soon, the legs became weaker, so that he tired easily, was unsteady on his feet and staggered when he walked. The pain was located in the lumbar and sacral regions, was worse on sitting than on standing and was accentuated by coughing, sneezing and straining. For four months the patient had had difficulty in starting the urinary stream. The bowels had become constipated.

Examination.—The patient was a well developed and a healthy looking man. He was unsteady when he attempted to walk, with a "steppage" gait. There was some weakness of the left lower extremity and of the hamstring muscles on the right side. The power to flex the foot dorsally was lost on the left, but was retained on the right side. There was some atrophy of the muscles of the right leg, with fibrillations in the muscles of both calves. The abdominal reflexes were present, but none of the tendon reflexes in the lower limbs could be elicited. Cutaneous sensibility was diminished below the second lumbar dermatome, more on the left than on the right side.

Manometric tests showed an almost complete subarachnoid block; the fluid was yellow and only a small amount—insufficient for examination—could be withdrawn.

Operation.—On Sept. 2, 1928, a large intradural growth, at least 6 cm. in length, was found which had pushed the roots of the cauda equina to the left. The tumor was extremely vascular, and was only partially removed. The pathologic report was "unclassified" giant tumor of the cauda equina.

CASE 3.—*Symptoms of two years' duration; exploratory laminectomy; inflammatory lesion of the roots of the cauda equina.*

History.—E. W., a married woman, aged 58, had always been well except for injuries sustained in a fall in 1921 and an attack of influenza in December, 1927. The patient was admitted to the Neurological Institute on Sept. 14, 1928, with the complaint of progressive loss of power in the left lower extremity. In 1926, she injured her left ankle, and following this noticed that she could not stand on the toes of the left foot. The weakness gradually progressed and involved the leg and then the thigh. During the winter of 1927 and 1928, the left foot and leg seemed colder than the right, and in April, 1928, she began to experience a sensation of numbness along the outer side of the left thigh and leg. She had no pain,

either spontaneous or on coughing and sneezing. Since July, 1928, she had had occasional urinary incontinence.

Physical Examination.—The patient was a fairly well built, well developed and well nourished woman who did not appear ill. The muscles of the left leg and thigh were flaccid; the left leg felt cold and was somewhat cyanosed below the knee. When she walked she dragged the left extremity, and there was a definite drop-foot on the left. No fibrillations were noted at the time of the examination. The abdominal reflexes were present and equal. The deep reflexes in the lower extremities were more active on the right than on the left, and the left achilles reflex was lost. The left leg was paretic, the loss of power being greatest in the foot, in which flexion and extension were equally involved. There was slight atrophy of the muscles of the left leg. Sensory examination revealed an area with ill defined margins on the outer aspect of both calves, extending almost half

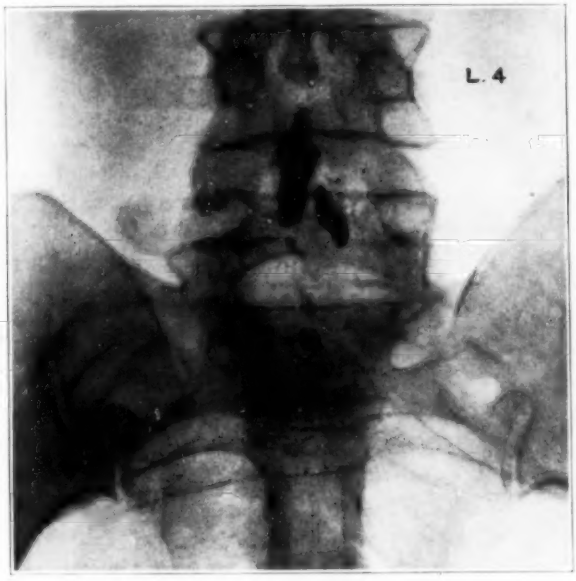

Fig. 3 (case 3).—Neuritis of the cauda equina, showing x-ray appearance of iodized oil arrested at the fifth lumbar vertebra.

way around the legs, in which sensibility for pain and temperature was lost. Muscle, joint and tendon and vibratory sensibilities were well preserved.

Roentgen examination of the lumbosacral region showed some evidence of early arthritic changes in the vertebrae. There was a bifid condition of the first sacral vertebra.

Lumbar puncture between the third and fourth lumbar spines showed a complete subarachnoid block. The spinal fluid was clear and colorless, contained 4 cells, no excess globulin and 23 mg. of protein. One week later, a second puncture was done between the fourth and fifth lumbar vertebrae. Again the fluid was clear and colorless, containing only 5 cells, no excess of globulin and 35 mg. of protein, and the manometric tests demonstrated a block in the subarachnoid space. Iodized oil was introduced into the spinal canal at the second puncture. The iodized oil remained at the level of the fifth lumbar vertebra; only a very small drop descended to the level of the first sacral vertebra (fig. 3).

Operation.—In spite of the fact that the low protein content of the spinal fluid made a tumor improbable, an exploratory laminectomy was performed. The arches of the third, fourth and fifth lumbar vertebrae were removed. The dura was rather bluish, but no resistance was felt. The arachnoid appeared cloudy and had several small white plaques in its substance. Two large calcareous plaques, 8 by 5 cm., were found attached to the overlying dura. A diagnosis of an inflammatory lesion with arachnoid adhesions was made.

CASE 4.—*Symptoms of eight months' duration; laminectomy revealed inflammatory changes in the roots of the cauda equina.*

History.—J. G, a man. aged 49, was admitted to the Neurological Institute in March, 1928, complaining of pain in the left lower extremity associated with pain and slight diminution in size of the affected leg. This pain had first appeared in August, 1927, before which time he had always been well. The pain affected the

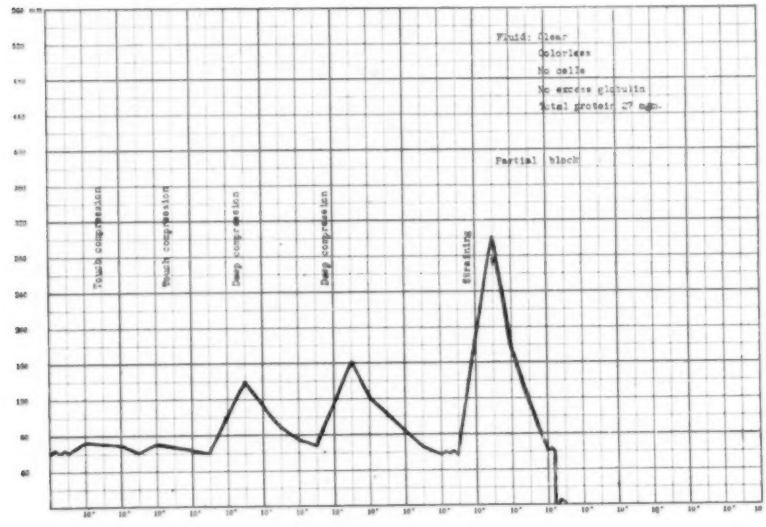

Fig. 4 (case 4).—Neuritis of the cauda equina. Manometric chart showing partial subarachnoid block.

lower third of the leg on its outer surface, was made worse by walking and usually subsided after a night's rest. The pain became steadily worse—both in intensity and in distribution—so that by November, 1927, it had involved the leg and the outer side of the knee. By January, it had extended to the outer surface of the thigh, and by February to the hip and left buttock. He found it impossible to lie on the left side because of the accentuation of pain in this position. Coughing, sneezing and straining at stool would precipitate and aggravate the pain. About six months after the onset of symptoms he was forced to discontinue work. About December, 1927, the patient noted that sensation was diminished in the first, second and third toes of the left foot. The loss of feeling gradually extended until the outer surfaces of the leg and thigh were affected. For two years, the patient had frequency and urgency of urination.

Physical Examination.—The patient was well nourished. Although he walked without difficulty, he was able to stand less well on his left than on his right foot.

The abdominal reflexes were easily exhaustible on the left and very weak, if at all present, on the right. The deep reflexes in the lower extremities were diminished, but less active on the right than on the left. There was some impairment of muscular power in the extensors of the left leg, adductors and flexors of the left thigh and in both dorsal and plantar flexors of the left foot. There was some

Fig. 5 (case 4).—Roentgenogram of iodized oil arrested at the second lumbar vertebra.

atrophy of the muscles of the left leg and thigh and of the left glutei. Sensory examination revealed some indefinitely defined hypesthesia and hypalgesia over the left thigh and calf of the leg, in the first and second sacral dermatomes. There was marked tenderness along the course of the left sciatic and external popliteal nerves. The spinous processes of the second and third lumbar vertebrae were slightly sensitive to pressure. X-ray pictures of the lumbosacral region

showed a slight left scoliosis; the fifth lumbar vertebra was almost completely sacralized on the right.

A manometric test showed a partial block in the subarachnoid space. There was a delayed fall on touch compression and a limited rise and delayed fall on deep compression with no measurable new level after the removal of 7.5 cc. of spinal fluid. The fluid, however, was clear and colorless. It contained no cells, no excess globulin and only 27 mg. of protein. Iodized oil was injected and descended only as far as the second lumbar vertebra (figs. 4 and 5). A preoperative diagnosis was made of inflammatory disease of the roots of the cauda equina with the possibility of tumor of the cauda equina.

Operation.—An exploratory laminectomy was performed on March 30, 1928. The arches of the first and second lumbar vertebrae were removed. The caudal roots were considerably congested; there was a moderate degree of neuritis of

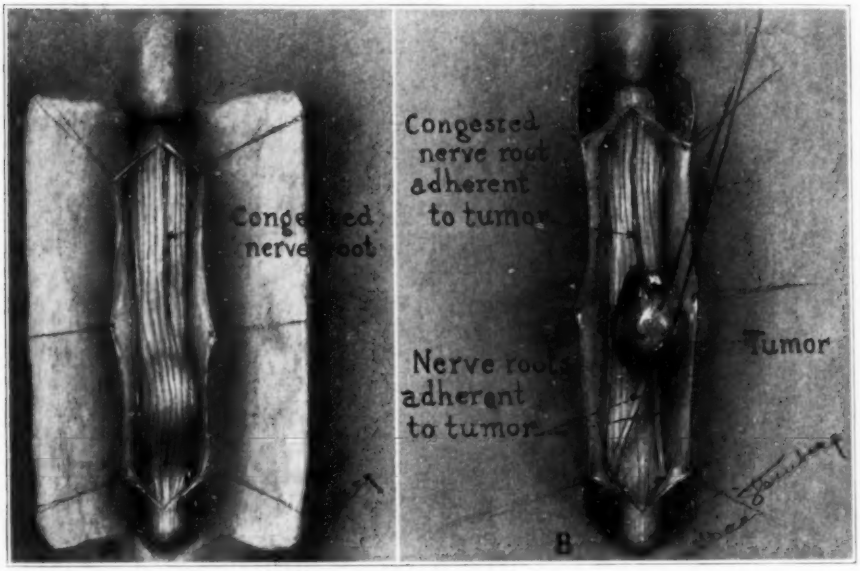

Fig. 6 (case B. S.).—*A*, backward dislocation of the roots of the cauda equina by a small tumor. *B*, a small tumor (perineural fibroblastoma) of the cauda equina.

the cauda equina, but no evidence of an extradural or intradural neoplasm. After the operation, the pain from which the patient had suffered subsided to a great extent, and he was discharged nineteen days after the operation much improved as far as the pain was concerned, although the neurologic status remained unchanged.

These four clinical histories represent the characteristic story told by the patients—a beginning with pain in the small of the back or down one lower extremity, followed by increasing weakness in one or both lower limbs and more especially of the power of dorsal flexion of the feet, sensory disturbances over the buttocks, the feet and especially the

outer aspects of the legs and thighs, and finally disturbances in the functions of the bladder and the bowels.

COMMENT

Individual Growths.—The size of intradural tumors of the cauda equina varies greatly. The growths of from 1 to 3 cm.—well encapsulated meningeal and perineural fibroblastomas (fig. 6)—met with at other levels of the spinal cord, are frequent also between the roots of

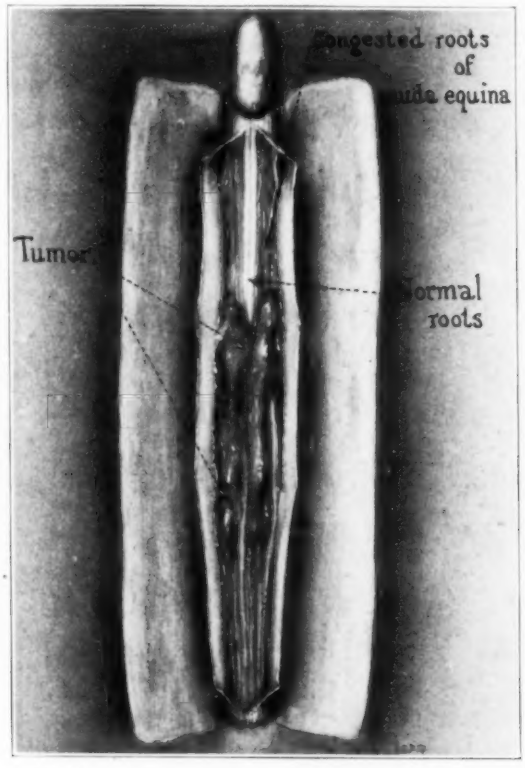

Fig. 7 (case G. W.).—A tumor of the cauda equina which enveloped the caudal roots.

the cauda equina. Growths which envelop the caudal roots (fig. 7) or push the roots to one side and fill the greater part of the lower end of the vertebral canal are the largest intradural spinal tumors encountered. The histology of these large tumors varies greatly and in not a few instances it has been impossible, thus far, to classify them satisfactorily. Because of the large size they attain, we have called them "giant" tumors.

These large growths are rarely adherent to the dura and probably develop within the arachnoid. They are usually encapsulated, although the membrane which surrounds them is very thin and easily ruptured during operative manipulations. The dura is often much thinned and stretched and the vertebral canal distinctly enlarged (fig. 8 and 9). Most of the tumors are poorly supplied with blood vessels, so that little bleeding is encountered during their total or fragmentary removal, but occasionally one meets with a large growth which is extremely vascular.

Extradural Tumors.—Tumors of this location that compress the roots of the cauda equina, usually derived from the bone or interverte-

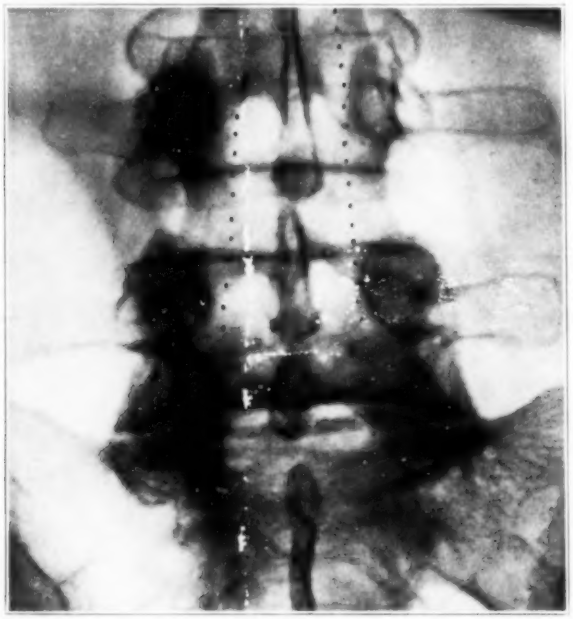

Fig. 8.—Roentgenogram of the lower lumbar vertebrae, to show the width (dotted) of the normal vertebral canal.

bral cartilages, are relatively infrequent. Twenty-three of the twenty-eight growths of the cauda equina in this series were intradural and four extradural; one was both inside and outside the dural sac. The growths outside the dura were chondromas (three of our series), sarcomas (one of our series) or chordomas (one case).

Histology.—Histologically, the tumors were classified as shown in table 1.

Incidence.—Males and females were about equally represented—fourteen males and fourteen females had tumors, and nine males and eight females had neuritis.

The average age was slightly less for the tumor than for the neuritis group—32 years for the former as against 40 years for the latter. As shown in table 2, 61 per cent of the patients with new growths were between 20 and 40, while 65 per cent of those with neuritis were between 40 and 50 years of age.

TABLE 1.—*Histologic Classification of Tumors*

	Extradural	Intradural
Sarcoma	1	5
Chondroma	3	0
Meningeal fibroblastoma	0	3
Perineural fibroblastoma	0	2
Spongioblastoma	0	2
Ependymal glioma	0	1
Medulloblastoma	0	1
Angioma	0	1
Chordoma (extradural and intradural)	1	0
Unclassified	0	6
	5	21

TABLE 2.—*Frequency According to Age*

	Tumors		Neuritis	
10 to 20 years	4		0	
20 to 30 years	9	61%	2	
30 to 40 years	8		3	
40 to 50 years	5	25%	11	71%
50 to 60 years	2		1	
Totals	28		17	

TABLE 3.—*Duration of Symptoms*

	Tumors	Neuritis
Less than 6 months	5	5
6 to 12 months	6	1
1 to 2 years	6	3
2 to 3 years	5	3
3 to 4 years	3	2
More than 4 years	3	1
Totals	28	15

These figures show that neuritis of the cauda equina is more frequent after the fortieth year (71 per cent), while new growths are more often encountered in patients less than 40 years of age (75 per cent).

The average time that elapsed from the onset of symptoms until the patient's admission to the hospital was less for the neuritis group—twenty months as compared with thirty months for the tumor group.

In three of the patients with tumors there was a history of trauma. Two patients had sustained an injury to the back, one ten months and

the other nine years before the onset of symptoms. In a third case, occasional backache became continuous after a strain of the back caused by the lifting of a heavy weight.

One of the patients with neuritis gave a history of trauma to the back eight months before and another injury to the back during diving.

Symptomatology.—As might have been expected from the anatomic location of the lesions, pain was the chief complaint that led the patients of both groups to seek medical aid.

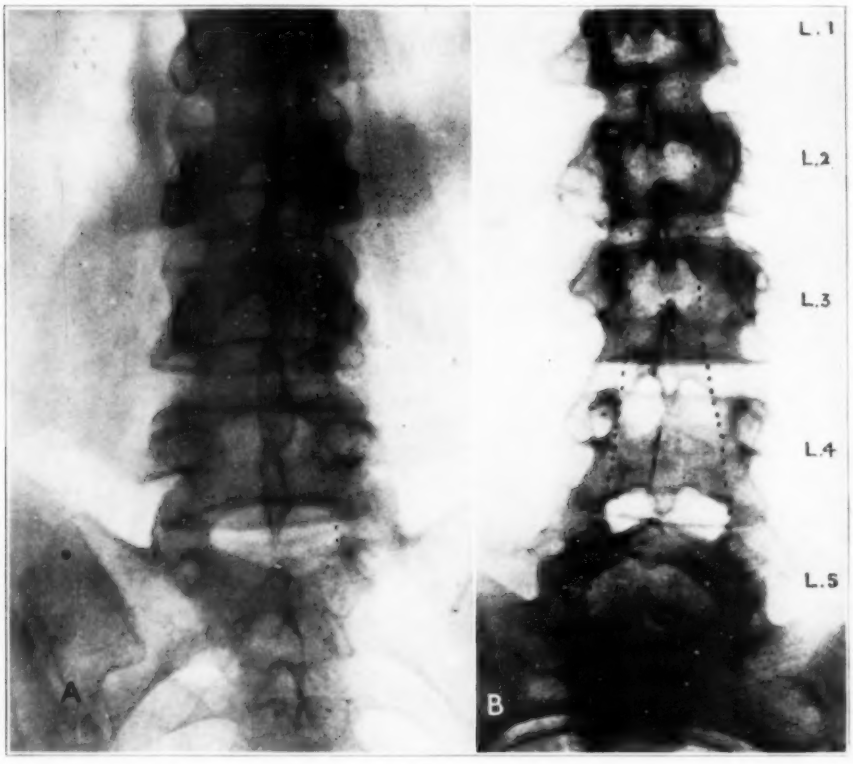

Fig. 9.—*A*, roentgenogram, showing enlargement of the lumbar vertebral canal in a case of large tumor of the cauda equina; *B*, enlargement of the vertebral canal in the fourth and fifth lumbar vertebrae in a patient (case 1) with a large caudal tumor.

All of the patients with tumor had pain at some period during the progress of the disease, while only two of those with neuritis were free from pain throughout. Weakness of both legs with sensations of coldness of the feet was the chief complaint of one of these two patients, while in the other it was progressive loss of power in one lower limb, with numbness and coldness in the leg and thigh.

In twenty-six of the twenty-eight patients with tumors and in fifteen of seventeen cases of neuritis, pain was the first symptom. Two patients with tumors first noted weakness of the lower limbs, and two with neuritis first complained of loss of power and subjective sensations of cold in the feet and legs.

In sixteen of the cases of tumor (57 per cent) the first pain was unilateral—in one limb in six, in the back and one limb in five, in one hip and lower limb in two and in one hip alone in three. Sooner or later, however, in all but one of the patients, the pain involved both limbs.

In twelve of the cases of neuritis (80 per cent), the pain first affected one lower extremity, and in almost one half of these it remained confined to one side. This is shown in table 4.

TABLE 4.—*The Location of the Pain*

	Tumor	Neuritis
Pain, unilateral at onset	16 (57 per cent)	12 (80 per cent)
Pain, remained unilateral	1 (4 per cent)	7 (47 per cent)
Pain, bilateral at onset	12 (43 per cent)	2 (13 per cent)

TABLE 5.—*Subjective Sensory Disturbances (Exclusive of Pain)*

	Tumor	Neuritis
Numbness	8 (29 per cent)	2 (12 per cent)
Paresthesias (tingling, pin and needle sensation)	5 (18 per cent)	..
Burning	4	3 (18 per cent)
Sense of weight	3	..
Coldness or warmth	1	2

The pain was often felt on the posterior surface of one thigh or down the entire extremity, and not rarely the patients had been treated for supposed "sciatica."

In about one fourth of the patients of each group, the pain was most severe in the middle of the lower part of the back. Pain in the back is a frequent complaint in patients both with tumors between the roots of the cauda equina and with inflammatory changes, and the story of the patient is often that of a "lumbago" of many months' or years' duration. Tenderness of spinous processes of the lumbar or sacral vertebrae was found in 65 per cent of patients with tumors as against 28 per cent of those with the neuritides; it was more often over the lower lumbar vertebrae and the sacrum in the tumors and over the upper lumbar vertebrae in the other group.

Exclusive of pain, subjective sensory disturbances occurred in 75 per cent of the cases of tumors and in 41 per cent of cases of neuritis, as shown in table 5.

Motor weakness, second in frequency among the symptoms, occurred in twenty-three of twenty-six of the tumor and in fifteen of seventeen

of the neuritis group. Bilateral loss of power occurred in three fourths of the patients with new growths and in three fifths of those with neuritis. The presence of drop-foot was specifically mentioned only in seven patients with tumors and in three with neuritis, but diminution of the power to flex the foot dorsally was frequent. Muscle atrophies were noted in fourteen of the former and in nine of the latter, and fibrillations in six as against three.

In tumors, the atrophy was most marked or entirely limited to the anterior tibial and peroneal groups of muscles, while in neuritis the wasting usually involved most or all of the musculature of the lower limbs.

Bladder Disturbances: These occurred in about 80 per cent of each group; they appeared rather more early in neuritis—on the average in ten months as compared with twenty months in the patients with tumors. Eighteen or 64 per cent of the persons with tumor had more or less retention, while six were incontinent. In the neuritis group, retention

TABLE 6.—*Frequency of Clinical Symptoms*

	Tumor		Neuritis	
	Present In	Absent In	Present In	Absent In
Pain	28 (100 per cent)	0	15 (88 per cent)	2
Motor weakness	23 (82 per cent)	5	16 (94 per cent)	1
Bladder disturbances	22 (82 per cent)	6	14 (82 per cent)	3
Subjective sensory disturbances	15 (54 per cent)	13	6 (35 per cent)	11

of urine requiring catheterization occurred in six (42 per cent), retention alternating with incontinence in three, and incontinence in four.

Sensory Disturbances: While alterations in cutaneous sensibility occurred in both diseases (85 per cent of tumors and 80 per cent of neuritis), the upper level of the changes was definite in all of the cases of tumor but vague in 40 per cent of cases of neuritis.

In both diseases, the areas over which sensation was disturbed were usually not the same in the two limbs, but this asymmetry was more distinct in the patients with tumor and could be more definitely outlined. There were, however, some striking exceptions to this rule. Occasionally, in the extremely large and soft tumors which were described many years ago by one of us as "giant tumors of the cauda equina," the alterations of cutaneous sensibility were so slight that without careful and repeated examinations they might have been missed altogether.

Reflex Disturbance: Diminution or loss of tendon reflexes in the lower extremities was much more often noted in tumors. In both diseases, suprapatellar and patellar reflexes were often diminished or not elicited, and the achilles reflex was usually less than normal or lost on one or both sides.

In tumors which are located entirely over the roots of the cauda equina and do not extend over the lower part of the lumbosacral cord, the abdominal reflexes are always unchanged. Diminution or loss of the lower abdominal reflexes in a patient in whom a tumor of the cauda equina is suspected always means that either the growth is very large and not limited to the caudal roots alone, or that the condition is inflammatory or degenerative and not a tumor at all.

Roentgenologic Changes.—These changes are occasionally observed and may be of considerable significance. No pathologic change due to the disease is ever observed in neuritis, but in "giant" tumors of the cauda equina the enlargement of the vertebral canal is sometimes shown very distinctly on the x-ray films (fig. 9). Therefore, the absence of these bony changes does not mean that there is no tumor, but a positive observation makes the diagnosis of a new growth certain.[2]

The Alterations in the Cerebrospinal Fluid; The Results of Manometric Tests; Double Lumbar Punctures. Iodized Oil.—In twenty-five

TABLE 7.—*Changes in the Tendon Reflexes of the Lower Limbs*

	Tumor	Neuritis
Absent, on one side	3	1
Absent, on both sides	12	1
Preserved	4	3
Exaggerated	0	4
Achilles reflex absent on one side	0	4
Achilles reflexes absent on both sides	5	3

of the cases of tumor and in twelve of the cases of neuritis, lumbar puncture was performed. In one of these no fluid was obtained, probably because the needle was inserted directly into the tumor. In another case, punctures were done successively below the fourth lumbar, fifth lumbar and first sacral spines with a negative result, but fluid could be obtained only from a puncture done in the first lumbar interspace.

Bloody fluid or pure blood escaped from the needle in two cases: At the operation, one patient was found to have a very vascular growth which completely filled the lumbar vertebral canal so that the tumor itself must have been punctured; in the second case, a distinct resistance was felt when the needle penetrated the vertebral canal.

Table 8 shows that in cases of tumor the fluid obtained by lumbar puncture in many instances shows the characteristic changes found in compression of the cord—xanthochromia, increase of globulin and of total protein without increase of cells—while in neuritis, xanthochromia

2. No reference, of course, is made to the arthritic changes so frequently seen in persons of or past middle age, to erosion of bone seen in primary or secondary malignant disease, or to the shadows cast by extradural osteomas or chondromas.

is unusual, increase of globulin not frequent, but increase of total protein never observed.

As is well known and therefore need only be mentioned, deep orange-yellow fluids are met with almost exclusively above or below tumors in the lowest parts of the vertebral canal; a chrome-yellow cerebrospinal fluid means tumor in the region of the conus or roots of the cauda equina. In compression of the cord at higher levels the color of the fluid is usually light yellow—similar to that which occurs in some inflammatory lesions, in tumors near or in the ventricles of the brain and as a final stage in intracranial and spinal hemorrhage from trauma or other cause.

TABLE 8.—*Alterations in the Cerebrospinal Fluid*

	Tumor	Neuritis
Colorless	6 (22 per cent)	8 (48 per cent)
Xanthochromia	15 (55 per cent)	1 (6 per cent)
Bloody	3	2
No fluid	1	0
Increase of cells	3 (215, 300, 40 cells)	1 (35 cells)
Increase of globulin	21 (87 per cent)	4 (50 per cent)
Coagulated on withdrawal	9 (37 per cent)	0
Increase of protein	21 (100 per cent)	0
Highest total protein	300 mg.	42 mg.
Average total protein	148 mg.	34 mg.

TABLE 9.—*Manometric Tests in Twelve Cases of Tumor and Nine of Neuritis of the Cauda Equina*

Subarachnoid Block	Tumors	Neuritis
Complete	5 (42 per cent)	3 (33 per cent)
Partial	2 (17 per cent)	1 (11 per cent)
Questionable	2 (17 per cent)	1 (11 per cent)
No block	3 (25 per cent)	4 (44 per cent)

Increase of cells was found, however, in three of the patients with large tumors, and there is considerable probability that in these cases, as a result of injury by the lumbar puncture needle, the cells were derived from the tumor itself.

The Manometric Tests.—As one would expect in the lower end of the vertebral canal where a tumor may be located entirely below the point at which the lumbar puncture is performed, only 59 per cent of the patients with tumors had a positive subarachnoid block, but it was surprising that 44 per cent of the cases of neuritis also showed partial or complete block.

In the five patients with tumors and a complete subarachnoid block, the globulin was between 1 -|- and 4 +, and the total protein was much increased; in the two patients with partial block, and in the two with doubtful block the protein content was always well above the normal, from 45 to 300 mg.

In the cases of neuritis with a positive block, on the other hand, increase of protein was never recorded.

The difference between the two groups—in the patients who had no evidence of block—was also striking: in all of the patients who had tumors the total protein was above normal, while in all of the patients with inflammatory changes of the caudal roots, the total protein content of the spinal fluid was within normal limits.

While the manometric tests are not conclusive in the differential diagnosis between tumor and neuritis, the estimation of the total protein content is of great value. In our series not a single instance of tumor occurred in which the amount of protein was not increased. Based on our experience, a diagnosis of tumor should rarely, if ever, be made unless there is at least some increase of globulin or of total protein.[3]

The Value of Double Punctures.[4]—In no region of the vertebral canal is a double puncture as useful for both diagnosis and determination of the level as in the lumbosacral areas.

TABLE 10.—*Double Punctures in Caudal Tumors*

	First Puncture			Second Puncture		
Vertebral Level of Tumor	Below Spine of	Block	Protein	Below Spine of	Block	Protein
1. L 2 to L 3	L 4	Partial	300 mg.	L 1	None	45 mg.
2. L 3 to L 5	L 3	Complete	.....	Th. XII	Partial	300 mg.
3. L 1 to L 5	L 3	Complete	No fluid	L 2	Complete	No fluid
4. L 4 to L 5	L 4, L 5, Sl	Complete	No fluid	L 1	Partial	75 mg.

Double punctures were performed in four of our patients with tumors, and the synopsis of our experiences in table 10 show the valuable data obtained in each case.

In three of the four patients, the evidence was conclusive that the upper limit of the tumor was below the level of the upper puncture, while in the fourth it was fair to conclude that the growth extended above the intervertebral space through which the needle was inserted. Thus the double punctures were of no little value for the localization of the growth in each instance. From the time that we had these experiences, we have regularly done double punctures for the localization of tumors of the cauda equina.

There were a number of patients in whom there was no block, but xanthochromia and increase of globulin or of total protein made the

3. We are cognizant of the fact that in the early stages of extradural tumors, there may be no increase of protein. We have seen this not so rarely in extradural compression of the spinal cord, but, up to the present time, we have not observed an extradural tumor in the region of the cauda equina with a normal amount of protein in the spinal fluid.

4. A paper on this subject will soon be published from our clinic by Dr. Cramer.

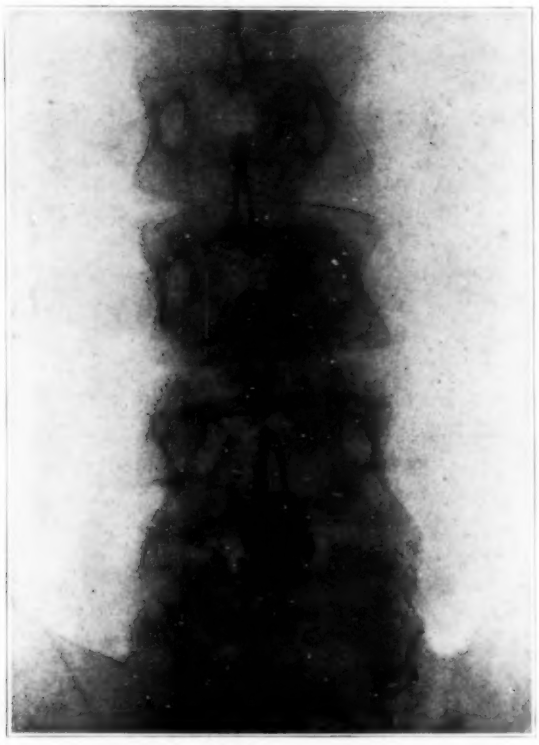

Fig. 10 (case E. H.).—Roentgenogram of iodized oil arrested at the fifth lumbar vertebra in a case of extradural chondroma below that level.

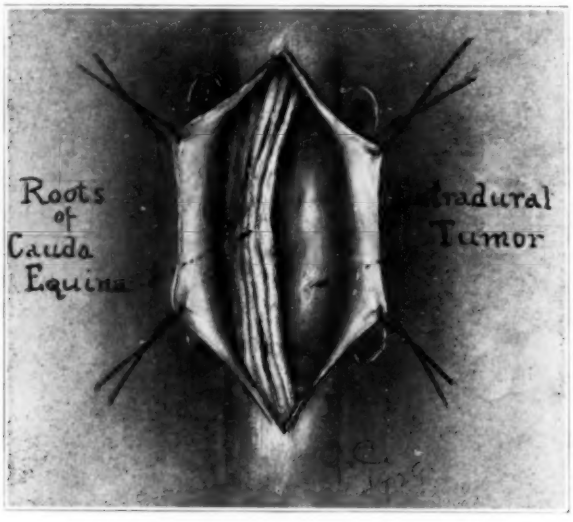

Fig. 11.—The condition found at operation in the case illustrated by figure 10.

diagnosis of "tumor" probable; the clinical signs left us uncertain, however, whether the growth was near the origin of roots from the cord or nearer the foramina of exit from the vertebral canal. Injections of iodized oil were made into some of these patients.

The Use of Iodized Oil in Suspected Tumors of the Cauda Equina.—In the surgical clinic of the New York Neurological Institute, we have taken a conservative attitude regarding the use of iodized oil, and we are convinced that—if thorough and repeated neurologic exami-

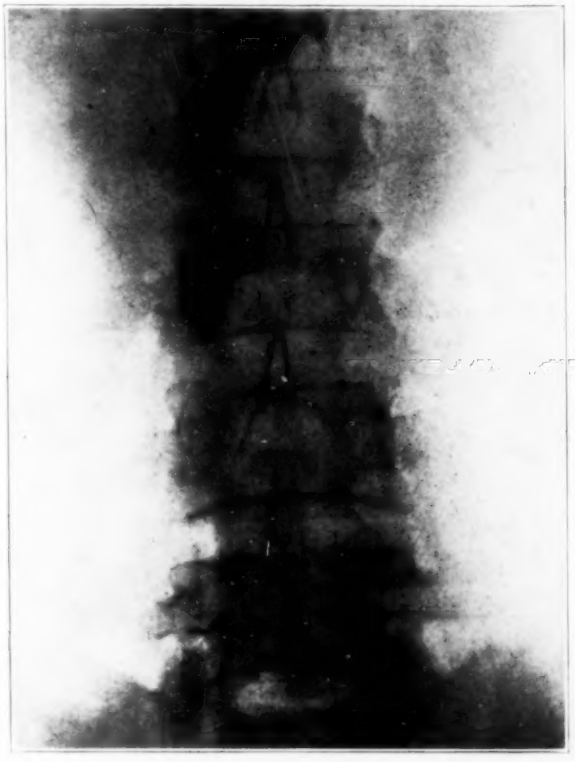

Fig. 12 (case M. T.).—Roentgenographic appearance of iodized oil arrested at the second lumbar level. Compare with figure 13.

nations and complete manometric tests have been made and the spinal fluid has been examined for total protein—injections of iodized oil are rarely, if ever, necessary for the diagnosis of growths that compress the spinal cord or the roots of the cauda equina.[5] For the determination

5. The objections to the indiscriminate use of iodized oil or any similar substance will be summarized in a paper which is soon to be published. The details regarding spinal manometric tests and the characteristic changes in the globulin and total protein content of the spinal fluid in compression of the spinal cord have been fully described in a number of publications from this clinic.

of the level of a spinal tumor when the motor and sensory evidence is indefinite or wanting, and in tumors in the lower parts of the vertebral canal when it is uncertain whether the growth is situated near the emergence of roots from the cord or near the exit of the roots from the vertebral canal, iodized oil has a definite field of usefulness (figs. 10, 11, 12 and 13).

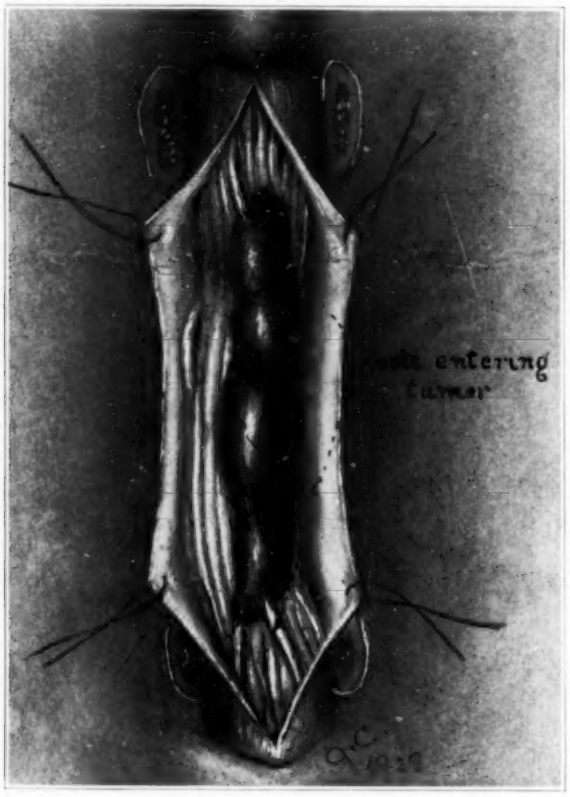

Fig. 13 (case M. T.).—The growth exposed at operation at the level of the iodized oil.

Injections of iodized oil were made into five patients with tumors and into three with inflammatory lesions of the cauda equina. In all the cases of tumor the information gained was of value, but in two the exact upper border of the new growth did not correspond to the shadow of iodized oil on the x-ray films.

In one patient (figs. 15 and 16) most of the iodized oil was arrested at the level of the lower border of the third lumbar vertebra, but the upper level of the tumor was at the fifth lumbar vertebra, and the

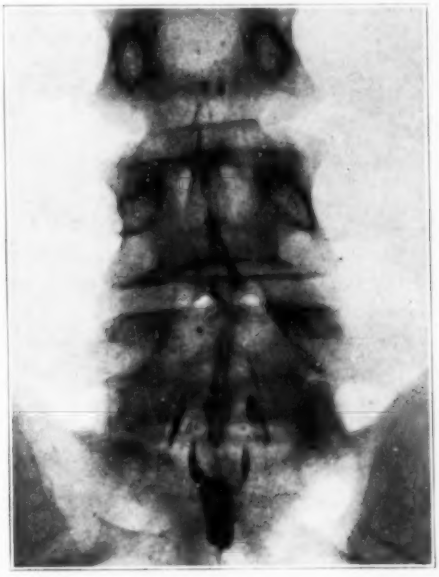

Fig. 14.—Neuritis of the cauda equina. Roentgenographic appearance of the iodized oil which has fallen to the bottom of the arachnoid sac.

Fig. 15 (case M. K.).—Tumor at the level of the fifth lumbar vertebra. Iodized oil arrested at the third lumbar vertebra. Compare with figure 16.

arrest of the iodized oil at a false tumor level was due to the backward dislocation of the roots of the cauda equina by the tumor.

In another patient (fig. 17), the iodized oil was arrested at the fourth lumbar vertebra, but the tumor extended up only slightly above the lower end of the arachnoid culdesac.

In two of the patients with inflammatory lesions of the roots of the cauda equina, one with a complete and the other with a partial subarachnoid block, the iodized oil was arrested in one instance at the

TABLE 11.—*Iodized Oil in Eight Cases of Lesions of the Roots of the Cauda Equina*

Nature of Lesion	Preoperative Diagnosis Based on Clinical Signs	Upper Vertebral Level Based on Iodized Oil	Upper Vertebral Level of Tumor Found at Operation	Remarks
1. Meningeal fibroblastoma	Tumor of cauda, unlocalized	L 3, to right	L 5, to right	Punctures at four levels (table 10, case 4)
2. Perineural fibroblastoma	Tumor of cauda, L 2 vertebra	L 2	L 2	Punctures at two levels (table 9, case 1)
3. Chondroma	Tumor of cauda, level ?	L 4, to right	L 4, to right	Lumbar puncture negative
4. Chordoma	Tumor of cauda involving especially 3 and 4 sacral roots on right	I 4	S 2, to right	Lumbar puncture negative
5. Chondroma	Tumor between L 3 and L 4 vertebra, to right	L 3-4, to right	L 3-4	Lumbar puncture negative
6. Neuritis with calcareous arachnoid plaques adherent to dura	Arachnoid adhesions, possibly tumor	L 5	.....	Complete subarachnoid block
7. Neuritis (fig. 14)	Neuritis of cauda roots	S 1-2	. ...	Lumbar puncture negative
8. Neuritis	Inflammatory lesion of roots, possibly tumor	L 3	.....	Lumbar puncture; partial block

level of the fifth lumbar vertebra and in the other instance at the level of the second lumbar vertebra (figs. 3 and 5).

The two cases of arrest of the iodized oil at a false tumor level are of no little importance. They demonstrate that—especially in the region of the cauda equina—the iodized oil may be arrested at a different level and by causes other than the tumor. We have had this occur in a third case, not included in this series, and believe that not so rarely adhesions between the roots of the cauda equina may cause the arrest of iodized oil above the actual location of the new growth.

On this basis, one can easily understand that in inflammatory processes of the caudal roots, the iodized oil may fail to fall to the bottom of the arachnoid sac, and may therefore give rise to the suspicion that there is a block due to a tumor. This occurred in two of our

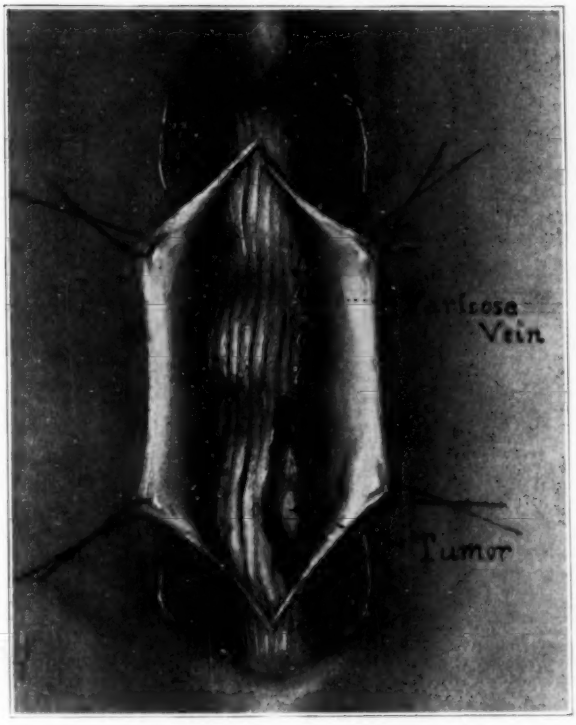

Fig. 16 (case M. K.).—The tumor exposed at operation.

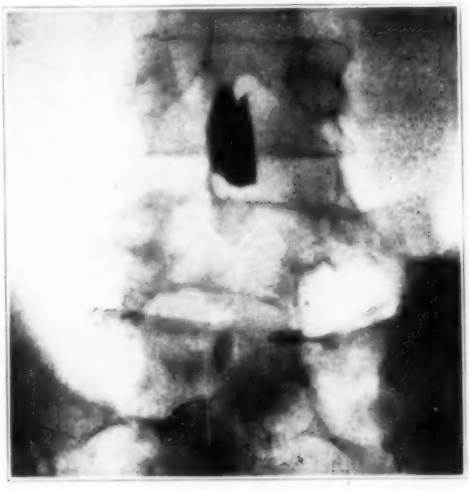

Fig. 17 (case L. S.).—Iodized oil arrested at fourth lumbar vertebra, in a patient with a tumor at the lower end of the arachnoid sac.

cases (table 11) and led to an exploratory operation in spite of the fact that the total protein in the fluid obtained by lumbar puncture was not increased above the normal.

SUMMARY AND CONCLUSIONS

Excluding secondary malignant disease of the vertebrae, tumors of the cauda equina form, in our experience, about 15 per cent of all extradural and intradural growths. In the majority of instances the questions of diagnosis that may arise in the study of symptoms and signs of the spinal cord and in the deductions to be drawn from the discovery of a partial or complete subarachnoid block are similar to those encountered in tumors at other levels of the spinal cord.

The absence of any change from the normal in the manometric tests of the spinal fluid is more frequent in tumors which compress the roots of the cauda equina because, in many instances, the upper end of the growth is caudad to the situation where the spinal puncture is performed. As a general rule, therefore, in suspected tumors in this region, the first puncture should be done as near the lower end of the arachnoid sac as possible, either in the fifth lumbar or even in the first sacral interspace. If no fluid is obtained then, punctures at one or two higher levels—below the third or fourth and the first or second lumbar spinous processes—should be performed.

The question whether a lesion is neoplastic or inflammatory may present serious difficulties. The main facts of significance in the cases we have studied were the following:

	Tumor	*Neuritis*
Age	Frequent before fortieth year	Frequent after fortieth year
History	Onset more gradual	Progress more rapid
Tenderness of spines	More frequent (65 per cent)	Less frequent (28 per cent)
Location of tenderness	More often over lower lumbar and upper sacral spines	More frequent over upper lumbar spines
Upper level of objective sensory disturbance	Definite in most cases	Vague in 40 per cent of patients
Spinal Fluid		
Xanthochromia	Frequent	Unusual
Coagulation	Frequent	Rare
Globulin	Increase in 80 per cent	Increase in 4 per cent
Total protein	Increase in all patients whether there was subarachnoid block or not	No increase whether there was a block or not
X-ray pictures	May show enlargement of vertebral canal	Always negative

If these facts are kept in mind, it should be possible, in all but exceptional instances, to arrive at a correct diagnosis, and we believe that in the future we shall not in our clinic do as many exploratory laminectomies as we have done in the past.

More difficult than the diagnosis is the exact localization of the growth from the standpoint of the laminectomy that is to be done. For this purpose, punctures at several levels and, when not conclusive, an injection of iodized oil through a needle in the first lumbar interspace, are usually of value. As we have pointed out, however, even the iodized oil may be held at a level which does not correspond to the exact upper or lower border of a tumor. Dislocated roots of the cauda equina, adhesions or calcareous plaques in the arachnoid may arrest the oil at some distance from the growth. In tumors which compress the lowest sacral roots near the sacral foramina below the ending of the arachnoid sac (of which we have recently seen an example) the iodized oil would fall to the bottom of the subarachnoid space, and unless the diagnosis of tumor was so certain that the fact of absence of obstruction in the subarachnoid sac would point to a growth at a still lower level, the injection of iodized oil might lead to the inference that there was no tumor.

When all of these facts have been taken into account, we are led to the conclusion that, as in growths of the spinal cord at other levels, valuable as other laboratory tests may be, the main reliance for the diagnosis of tumor of the cauda equina must be placed on a well taken history, careful and repeated neurologic examinations and thorough investigation of the pressure relations and the physical and chemical changes in the spinal fluid.

ABSTRACT OF DISCUSSION

DR. ERNEST SACHS, St. Louis: It was shown years ago by Dr. Ayer that iodized oil definitely increases the cell count in the spinal fluid and produces an irritative inflammation. Is it possible that the discoloration might not be due to the presence of the iodized oil?

DR. ISRAEL STRAUSS, New York: Was there a laminectomy in these cases of radiculitis?

DR. C. A. ELSBERG: Yes.

DR. ISRAEL STRAUSS: My reason for asking that question is this: In these cases of radiculitis, there are usually adhesions in the subarachnoid space interfering with the circulation, thus causing a block.

Granted this, if iodized oil shows a block, partial or otherwise, at any level in the spinal canal, it indicates, especially in cases of radiculitis, that there is or are adhesions. In patients with radiculitis with adhesions the conditions, as a rule, are not cured without laminectomy. So, what does it matter at what level the iodized oil shows the block, provided the entire region of the lumbar cord and of the cauda equina is exposed?

In one of the cases shown, the iodized oil showed a block a considerable distance above the tumor, the tumor being in the sacral portion of the canal. We have no explanation to offer for this, unless possibly there was some adhesion in the arachnoid which was overlooked, because those adhesions are sometimes so tenuous that they are easily destroyed.

In a paper that will be published shortly by Dr. Globus and myself, we have reviewed the use of iodized oil in about ninety cases, and we can state with definiteness that we have found no evidence at any time of an injury of a permanent nature to the spinal cord or to the roots.

The fact of the matter is—and we have observed these cases by roentgenography over a period of two years—that the iodized oil remains fluid, and if you depress such a patient you will see the iodized oil running upward in the spinal canal. There is no fixation of it. Likewise, there is very little absorption.

Dr. Elsberg, in giving us this diagnostic differential point between tumors and radiculitis in the increase of the protein, has contributed something to our diagnostic methods that is of tremendous value. I feel, however, that in the proper cases, iodized oil may still be of service. And it is also important to warn those who use it that it is not sufficient at times merely to examine the patient immediately after the injection of iodized oil and twenty-four hours later, but that sometimes it is important to observe the position of the iodized oil for from two to six days following its injection. In such cases, for long observation, if the iodized oil still persists at one point, you may be absolutely certain that there is some obstructive lesion in the subarachnoid space at that point.

Dr. Wilder Penfield, Montreal: Through the kindness of Dr. Elsberg I have seen some of the so-called giant tumors of the cauda equina but have not made a complete study of them as yet. A certain number of these neoplasms fall, obviously, into a group that Bailey and Cushing have called "unipolar spongioblastoma," containing elongated cells with long tails, rather fibrous in appearance. There are others, however, which are very cellular and these I should be unwilling to classify until I have seen further stains.

Dr. Elsberg: No matter where the tumor is located, whether or not it is low—a number of vertebrae below where the puncture was done and the fluid obtained—there is always an increase of protein. There may be rare cases in which a small tumor situated low in the canal will not cause an increase of total protein, up to the present—based on our experiences at the Neurological Institute—there has not been a single case without an increase of protein. Therefore we do not feel justified in making a diagnosis of tumor unless there is increased protein, no matter whether the tumor is well below the location of the puncture or not.

Finally, regarding the remarks of Dr. Strauss, I want to say that the question whether iodized oil should be used or not was not the question that I wanted to stress. Iodized oil may have its indications, and undoubtedly it has.

I am, personally, averse to using, except when absolutely necessary, a substance that, to use Dr. Strauss' own words, will remain in the subarachnoid space for several years and flow up and down (up when the person lies down at night, and down when he gets up the next morning). I think that it most be granted that this cannot do any good, that it only can do harm.

In the inflammatory lesions of the cauda equina either the arachnoid adhesions or the swollen roots must be the causative factors in the block that one may find. I do think that it is of great importance to distinguish between new growths and inflammatory lesions, because I am not at all convinced, in spite of an occasional improvement after operation, that one should ever do a laminectomy for so-called

neuritis or radiculitis of the cauda equina if one can make the diagnosis beforehand. A certain number of these patients recover satisfactorily without operation. The question usually has been, "Is this a tumor, or not," and because no satisfactory conclusion could be reached, the laminectomy was done. If one makes an out-and-out diagnosis of an inflammatory process of the roots of the cauda equina, then one should not operate. The studies of Dr. Constable do show how one can make a differential diagnosis, how one can localize the disease and, therefore, pick out the patients who should be subjected to operation, and avoid surgical intervention in those in whom operation will probably do no good.

LEPTOMENINGITIS

FORMATION OF MACROPHAGES FROM ARACHNOID CELLS *

THEODORE T. STONE, M.D.
CHICAGO

The arachnoid or intermediate membrane consists essentially of an interlacement of flattened bundles of fine fibrous tissue interspersed with elastic fibers and platelike cells. In addition to the main sheath or the partition, both sides of which are covered with endothelium, numerous trabeculae extend across the subarachnoid space; in places they are so plentiful as to convert the cleft into a spongelike structure. Morphologically, these covering cells of the leptomeninges are low and flat with large, pale, oval nuclei. The chromatin net in the nucleus as a rule is indistinctly made out. With ordinary histologic stains the cell boundaries are not visible, but definite intercellular lines may be demonstrated by silver nitrate (Key and Retzius,[1] and Essick [2]). Fatlike material in the arachnoid cells was described by Key and Retzius.

The purpose in this study was to determine the origin of the meningeal cellular elements, primarily the macrophages, in early cases of leptomeningitis. It will be shown that the cells lining the subarachnoid cavity in man have the function of forming macrophages in response to the stimulus of an acute inflammatory process.

The available literature on this possible function of the arachnoid is scanty. Numerous contributions have been made, however, relative to the morphology of the cellular changes in acute leptomeningitis. These primarily deal with the type of cells and do not attempt to discuss their origin. Hassin,[3] in 1918, reported on the cellular changes in acute leptomeningitis. He studied purulent, pneumococcal, epidemic cerebrospinal, and tuberculous meningitis. He stated that in all of the aforesaid types of meningitis, plasma cells, macrophages and lymphocytes are found constantly. He differentiated the various types of acute

* Submitted for publication, March 1, 1929.

* Read at a meeting of the Chicago Neurological Society, Feb. 21, 1929.

* From the Division of Neuropathology, Department of Nervous and Mental Diseases, Northwestern University Medical School.

1. Key, G., and Retzius, A.: Anatomie des Nervensystems und des Bindesgewebe, Stockholm, 1875, vol. 2, 1876.

2. Essick, C. R.: Formation of Macrophages by Cells Lining the Arachnoid Cavity in Response to the Stimulus of Particulate Matter, Contributions to Embryology, 42, Carnegie Inst. Washington, 1920, no. 272, p. 377.

3. Hassin, G. B.: Cellular Changes in Various Forms of Acute Leptomeningitis, M. Rec. **93**:760 (May 4) 1918.

leptomeningitis by the types of cells present. Spielmeyer [4] stated that under the influence of inflammation, hemorrhages and subpial softenings, the leptomeninges show proliferation, and large cells are formed which have the power of devouring other cells. These cells originate from the fibroblastic network of the arachnoid and pia. Metchnikoff [5] divided the devouring cell into two groups: microphages and macrophages. The former are the polymorphonuclear leukocytes, and the latter can arise from various kinds of elements; for example, from the reticulo-endothelial cells, cells of serous membranes, blood vessel endothelial cells, alveolar endothelial cells, neuroglia cells, lymphocytes and mononuclear leukocytes. Maximow [6] stated that macrophages, like plasma cells, originate from the blood elements; namely, lymphocytes and mononuclear leukocytes. The exact method of transition was not

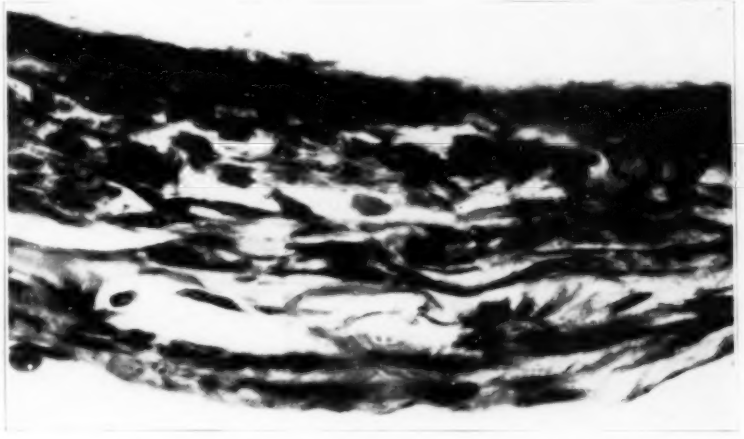

Fig. 1.—Thickened arachnoid with enlargement of arachnoid cells, Van Gieson; × 375.

described, but he did not believe that macrophages could take origin from the meninges, that is, from the arachnoid.

Experimental studies on the leptomeninges have led to other conclusions. As the result of his experimental work on the cells of the arachnoidea of the cat, Weed [7] drew the following conclusions: The

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arachnoid mesothelial cells are normally of a low flat type, but their morphology depends on the particular physiologic state of the cells at the time of examination. Under the stimulus of particulate matter and in acute infections the cells increase in size, become phagocytic and at times form freely moving macrophages.

In many early acute cases of experimental meningitis, Ayer [8] observed that the cellular exudation in the meninges was of the large mononuclear type. From a study of transitional stages in the cells still attached to the membrane he concluded that at least some of these cells represented macrophages derived from the arachnoidea. In the

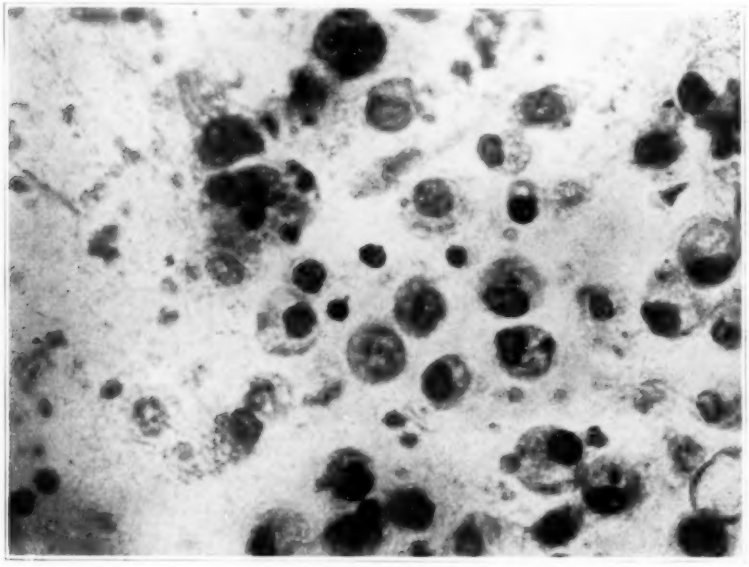

Fig. 2.—Macrophages in subarachnoid space in acute septic leptomeningitis. Toluidine blue; × 750.

presence of the toxins of such early infections, the mesothelial cells became enlarged with accumulation of the cytoplasm about the nuclei; the cell attachments became less extensive, and in some sections the cells appeared as if ready to be detached.

Essick [2] reached the following conclusions in his study of the cells lining the subarachnoid space: Particulate matter in the subarachnoid space causes mesothelial cells to accumulate and bud off from their attachments. These cells comply with the criteria for the classification of the macrophages; that is, they are ameboid and remove débris.

8. Ayer, J. B.: A Pathological Study of Experimental Meningitis from Subarachnoid Inoculation, Monographs of the Rockefeller Institute, 12, 1920, p. 26.

Normally these cells are found in the cerebrospinal fluid (mononuclears).

MATERIAL STUDIED

Twelve cases were studied in regard to the origin of the various cellular elements, chiefly macrophages. Epidemic cerebrospinal men-

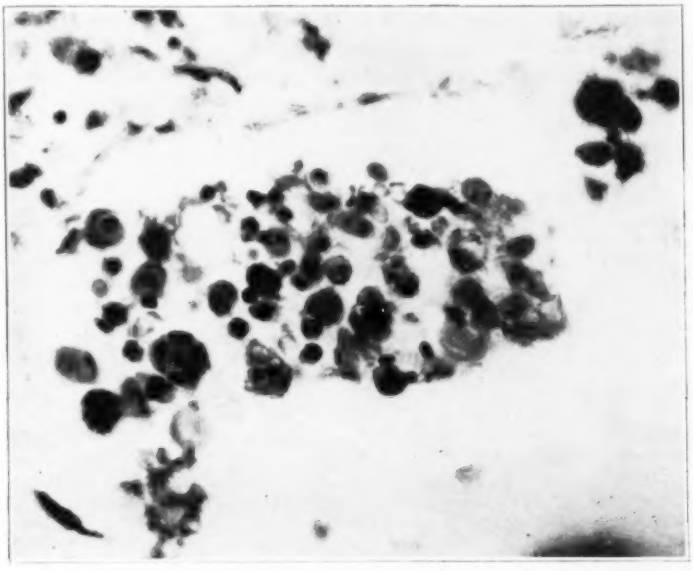

Fig. 3.—Macrophages in subarachnoid space in acute meningococcic leptomeningitis. The arachnoid (above) shows many swollen cells. Hematoxylin and eosin; × 520.

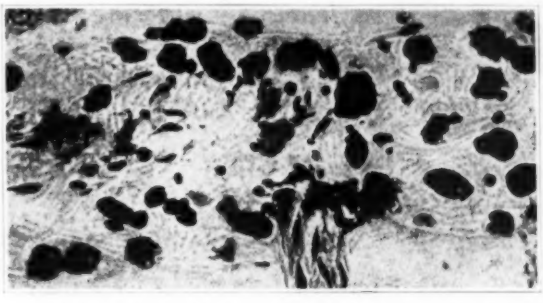

Fig. 4.—Macrophages filled with fat material in acute purulent leptomeningitis. Osmic acid; × 480.

ingitis was present in three cases, septic or purulent meningitis in one, tuberculous meningitis in six and syphilitic meningitis in two.

Methods.—Meninges from the convexity of the brain, from the base of the brain and from the pial septum of the brain were studied. All tissues were

hardened and fixed in 10 per cent commercial formaldehyde and stained with various stains—hematoxylin and eosin, toluidine blue, osmic acid, Unna's stain, Van Gieson's stain and silver nitrate. A detailed account of the complete microscopic structure in these types of meningitis will not be given here, as the picture presented by cases of this kind has been ably described before.

Epidemic Cerebrospinal Meningitis.—The pia was greatly infiltrated with plasma cells, lymphocytes, polyblasts, macrophages and polymorphonuclear leukocytes. Fibroblasts and gitter cells were likewise found. Plasma cells predominated in the inner zone of the pia, that portion adjacent to the brain. Polymorphonuclear leukocytes were present in only small numbers. Certainly there were more mononuclear leukocytes and small lymphocytes than polymorphonuclear leukocytes.

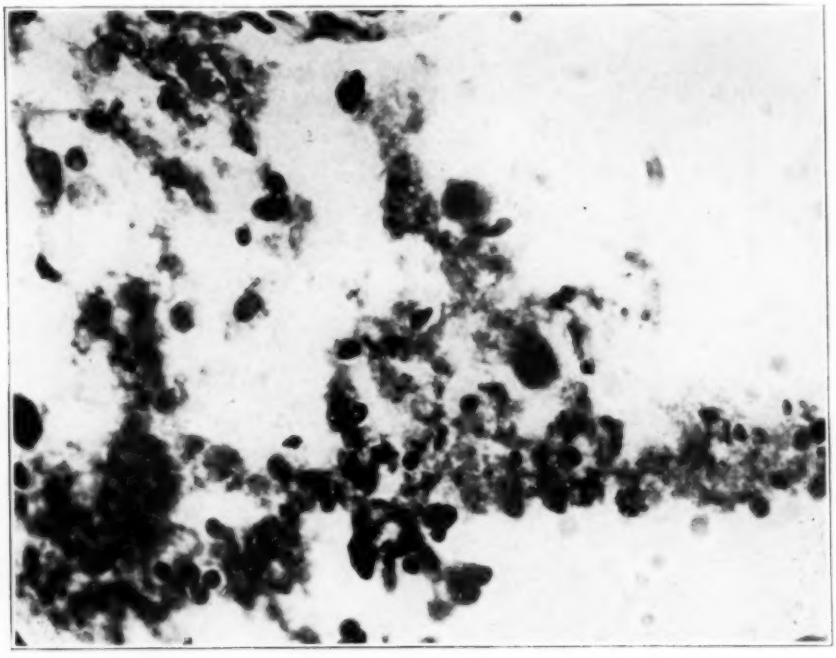

Fig. 5.—Syphilitic leptomeningitis. Many round cells with an occasional macrophage are seen. Plasma cells are numerous. Toluidine blue; × 520.

The pial septums showed many mononuclear leukocytes and plasma cells. In these three cases a close study was made of the arachnoid. In several areas on the convex surface of the brain there were found sections of the arachnoid in which large, circular or oval cells appeared. These were attached to the inner surface of the arachnoid and contained an eccentrically placed nucleus with the cytoplasm containing substances that stained black with osmic acid (fat globules) and in many instances contained structures resembling cocci. In other areas there was an increase in the number of arachnoid cells, so that a definite arachnoid cluster could be made out similar to that reported by Weed. Many parts of the arachnoid were entirely devoid of cells. In these areas a careful search in the subarachnoid space revealed the presence of large cells with eccentric nuclei and reticulated cytoplasm. The latter contained inert bodies, fat globules,

organisms and blood cells. In some of these cells, the cytoplasm appeared vesicular, and there was also some lattice-like arrangement. In this area polymorphonuclear leukocytes and small lymphocytes were seen infrequently. Fibroblasts and plasma cells were seen with mononuclear leukocytes. In several instances the fixed arachnoid cells that had become enlarged showed many inclusions, consisting chiefly of fat particles and inert substances. In a few instances, arachnoid cells contained structures resembling cocci.

Septic Leptomeningitis.—In the one case studied, the pia-arachnoid was found to be greatly infiltrated with pus cells. Microscopically, there were pronounced cellular and vascular changes only. The former will be described here. The cellular elements consisted of polyblasts, polymorphonuclear leukocytes, plasma cells, macrophages and several Hortega cells (gitter cells). With the exception of the plasma cells, the macrophages were by far the most numerous of all the cells present in the subarachnoid space. These devouring cells were similar to those found in the cases of epidemic cerebrospinal meningitis and contained numerous large vacuoles which in turn enclosed pus cells, fat globules, inert bodies, pigment and organisms. Gitter cells were found around the blood vessels and in the meshes of the pia. In the pial meshes, and over several areas of the

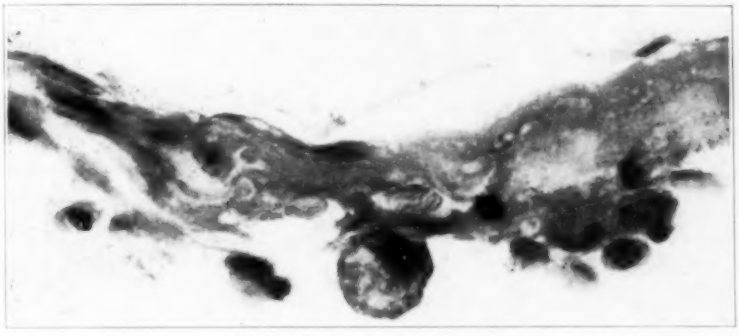

Fig. 6.—Formation of macrophage from arachnoid cell. Organisms are seen in the cytoplasm. Hematoxylin and eosin; × 850.

convexity and base of the brain, collections of pus cells were found. Few other types of cells were found in this region. In this case a detailed study revealed the presence of macrophages in the outer surface of the pia mater. In contrast to areas of the subarachnoid spaces in which no macrophages were found, when macrophages were numerous the arachnoid membrane was found to be devoid of cells. In other areas, the arachnoid cells were enlarged and almost ready to detach or break themselves away from the membrane. Most of these enlarged circular cells contained fat particles, blood elements and inert bodies.

Tuberculous Meningitis.—Six cases were studied in this series. Briefly, the pathologic observations were as follows: Macroscopically, apparent thickening of the pia mater of the base of the brain around the medulla, pons, chiasma and peduncular region with tubercle formation was seen; microscopically, the vessels were markedly increased in number with greatly hypertrophied and infiltrated walls. The cellular elements found in these cases were occasional macrophages, large numbers of plasma cells in various forms, fibroblasts and small mononuclear leukocytes. No polymorphonuclear leukocytes were found in any of these cases. A detailed study was made of the arachnoid and pia. In none of these cases was

there any evidence of enlargement of the normal arachnoid cells. There were no inclusions in any of the cells lining the arachnoid membrane. One subarachnoid space was particularly studied in relation to the arachnoid membrane and when considerable cellular exudation was seen there was no visible change in the cells of the arachnoid in either form, size or a tendency to detach themselves from the membrane. The few macrophages found contained no corpuscular elements or fat material.

Syphilitic Meningitis.—In one of the two cases studied pathologic changes were found in the meninges of both the convexity and the base of the brain, in the interpeduncular space in the region of the optic chiasm and about the oculomotor and optic nerves. In the second case pathologic changes were found only in the basal meninges in the interpeduncular space. Briefly, the changes were confined to the pia, producing an adherent pia-arachnoid with considerable thickening. Marked evidence of endarteritis was present in both cases. The cellular infiltration consisted of small lymphocytes and plasma cells, especially around the vessels. The latter process extended into the brain tissue proper. Marked proliferation of the connective tissue made it impossible to distinguish the pia and arachnoid in many parts of the basal meninges.

A detailed study in these two cases in regard to the arachnoid membrane showed no evidence of any enlargement of the arachnoid cells. No inclusions such as fat globules, organisms, red cells or inert bodies were seen. No macrophages were seen in the areas in which the normal arachnoid cell lining could not be made out. No evidence of detachment of any archanoid cells was noted in any of the sections studied.

COMMENT

In epidemic meningococcus and septic meningitides, it will be noted from the description given that the cells of the arachnoid participate in the formation of macrophages. According to Spielmeyer, the pathologic observations can be explained by the histogenetic changes of the nuclei of the arachnoid containing mesodermal nets. These nuclei play the most important rôle in the microscopic manifestation of disease of the meninges.

Under the influence of inflammations, hemorrhages and subpial softenings, there are proliferations which change the normal picture in two ways. Large cells arise which possess large bodies of protoplasm and which are related to nearby similarly progressively changed elements. Thus the nuclei which are often close together appear scattered in the syncytial masses. This is the so-called fibroblastic network. They constitute a fibrillary differential stage next to primitive mesenchymal filaments. The fibroblastic network under suitable conditions changes to collagenous fibrils. Under certain conditions, however, such as syphilis and tuberculosis, this change to collagenous fibrils does not take place, and the arachnoid remains in the fibroblast network stage. The macrophages of the epidemic and septic types of meningitis, in addition to having cellular enclosures, may be free from them, and one may be able to see a vesicular structure. The nucleus is small in relation to the size of the cell body and is pressed against the edge. The periphery

of the cell has a thickened lattice-like and spongy appearance. These properties are most prominent in those elements which have not devoured any cellular constituents, but in which the inner part appears light. It is to be assumed that in such elements the constituents previously taken up have become liquefied.

CONCLUSIONS

1. In early acute cases of leptomeningitis in man the cells lining the arachnoid and trabeculae may become macrophages.

2. The process of formation of macrophages in the arachnoid under the stimulus of purulent leptomeningitis consists of enlargement of cells, the eccentric displacement of the nucleus, and detachment of the cell which becomes ameboid and engulfs fat globules, inert bodies, cells and organisms.

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SOME NEUROLOGIC CONCEPTS APPLIED TO CATATONIA*

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It is a generally accepted theorem today that the nervous apparatus of higher animals is constructed on the plan of a pyramid of levels or systems through the erection of increasingly more complex organizations superimposed on the simpler, and that many of these levels probably indicate major phylogenetic progressions. Thus, one recognizes the parallelism between the asynaptic net of the mammalian intestines and that of *Medusa,* between the ganglionated cord of the sympathetic nervous system and that of the crustacea, between the spinal cord and the equipment of the amphioxus and between the midbrain centers of mammals and the "brain" of the lower fishes, birds and reptiles. In each animal the completed nervous structure of which represents one of these levels one, of course, finds a path of translation from the sensory or receptive mechanism to the motor or effector group. It is a principle of the pyramiding by which the more complex systems of the higher animals have been constructed that each lower level retains its structural integrity and, in part, retains its autonomy, but that the integration of its various inherent response patterns is controlled from higher centers. The implantation through which this higher level control is maintained takes place at the junction between the sensory and motor units, but apparently it merely subjugates the lower patterns and does not destroy the capacity for direct responses by way of an immediate path from the sensory to the motor mechanisms of these lower levels. Thus, one can recognize an important motor "cross over" at each phylogenetic level. In the asynaptic system this is inherent in the endless net structure. In the ganglionated cord it is constituted by the many neurons which serve as paths of communication between ganglia. In the spinal cord it is represented by the highly complicated group of propriospinal neurons which govern the many highly integrated reflex movements, such as the scratch reflex, which can be shown to be resident in the cord. At the level of the midbrain the "cross overs" form the important cerebellar, lenticular and rubral pathways.

Generally, the phylogenetic pyramid is carried one step into the brain proper in the recognition of important differences between the archi-

* Submitted for publication, May 1, 1929.

* Read at the Fifty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 28, 1929.

pallium and neopallium, and while, as yet, proof of clearcut evolutionary steps within this phylum has not been adduced in mammalian brains, there seems to be evidence that three fairly distinct levels exist within the neopallium of man. This rests on facts derived from cortical histology, myelogeny and clinical studies of differential functional losses and differential obstacles to acquisition of certain fairly precise cerebral functions such as speech and reading. That motor "cross overs" exist at each of these levels also is indicated on clinical grounds and they would seem to offer an explanation of certain otherwise bizarre action patterns which one often sees in catatonia.

Histologic studies of the cerebral cortex support the view of three progressive steps in the sensory cortices. The most precise demarcation of an anatomic field to be found anywhere in the cerebral mantle is in the area striata of the calcarine cortex which serves as the arrival platform for vision, i. e., the place of implantation of the relays from the pulvinar of the thalamus carrying optic stimuli. This cortical field is easily identified by the white line of Gennari (stria of Vicq d'Azyr) which is readily visible to the naked eye in the fresh brain. This cortex occupies a roughly pear-shaped area immediately surrounding the calcarine sulcus and its limits are abrupt so that even without the microscope one can clearly determine its boundaries. Surrounding this arrival platform on all sides, except the anterior or the stem end of the pear, is cortex of a different structural pattern. This is the area parastriata or common occipital type. As already stated, the inner borders of this field where it comes in contact with the calcarine type are sharply demarcated. Its outer borders are less easily determined. It comes into relation with the neopallium for a short distance on the mesial surface of the hemisphere, and here demarcation with the aid of the microscope is comparatively sharp. Where, however, it meets cortex of the temporal and parietal lobes its borders are not clearly differentiated and even with careful microscopic comparison it is difficult to assign to it an exact limit. When, however, sections from the middle of this field are carefully compared with similar sections from adjacent fields it becomes fairly obvious that one is dealing with structural patterns of different type.

I have chosen the visual cortices to illustrate these two levels since the visual field is the one to which much of my attention has been paid. Similar separation, however, can be demonstrated in the auditory field where the arrival platform is found to occupy the transverse temporal gyri of Heschl buried beneath the overhanging opercular convolutions in the fissure of Sylvius, and where the second type is found to occupy most of the first temporal gyrus. Two comparable fields are seen in

the postcentral gyrus and adjacent portions of the parietal field which probably subserve the epicritic fraction of spinal sensation. A similar classification of the olfactory and gustatory cortices is not as readily made because of less careful observations of the archipallial fields and a less thorough study of their relationships.

The third structural type of the posterior or the sensory division of the brain, while separable into many subtypes by detailed histologic comparison, in general conforms to a fairly common pattern. This occupies the large fields lying between the various second level types and hence covers the temporal, parietotemporal and parietal areas. It is of interest here to note that these third level fields are those which most clearly differentiate the sensory portions of the human brain from that of other primates.

Myelogenetic studies suggest three comparable developmental steps within the cortex. While there has been much contention concerning the relative rate of myelogenetic ripening of various areas of the cortex, in its general outlines Flechsig's original work seems fairly well substantiated. At or about the time of birth, the cortices which are mature in the sense of having received at least a considerable portion of their ultimate myelin content are limited strictly to the so-called projection areas which, in the sensory fields, consist of the arrival platforms. A second progressive step in myelinization occurs shortly thereafter when myelin appears in the areas of the second order, as stated by Flechsig. These are found immediately surrounding the projection zones or arrival platforms, and conform in general to the loci of the second histologic types. The third and last period is that when ripening occurs in the parietal and temporal zones.

From the evidence of both the structural differences and the myelogenetic periods one might anticipate the recognition of three more or less discrete elaborative levels in cerebral function. Under normal conditions these three are so intimately integrated that their separation is difficult. When functional disintegration occurs, however, an illuminating fractioning of sensory processes often takes place, which brings into fairly clear relief what appear as three clearly separate planes of elaboration. These were first noted in cases of losses due to cerebral injury or disease, and in the visual function correspond roughly to the clinical pictures of cortical blindness, mind-blindness and visual aphasia. The special educational disabilities, i. e., isolated failures in the acquisition of some function of the language faculty, yield evidence of great import concerning the third level, and studies of sensory acquisition of infants with the aim of correlating this process with the various functional levels would seem to offer much of promise concerning the first two.

THE THREE ELABORATIVE LEVELS OF CEREBRAL FUNCTION

My current envisagement of the function of the first elaborative level, which I tentatively relate to the arrival platform cortex, is that it supplies that fraction of the conscious experience which carries with it the conviction of external origin of a sensation and probably also yields exceedingly accurate information as to the position of the source of a stimulus with reference to the body. When this cortex alone is functioning, one meets the condition known as mind-blindness. That a motor "cross over" exists at this level is, I think, indicated by the fact that patients suffering with mind-blindness, in whom interpretation of the meaning of visual stimuli is entirely lacking, can yet find their way about without collision with obstacles in the environment. As a possible anatomic pathway for this "cross over" I should suggest the solitary cells of Meynert of the calcarine area. These are large cells of the motor type lying in a substellate position, and their axis cylinders are known to travel back toward lower centers from the calcarine cortex in the opposite direction to that of their companions in the optic radiations. Such a short cut can readily be conceived as a part of the mechanism whereby habituated movements are brought into immediate and accurate adjustment to the visual stimuli. In such activities as running over rough ground, fencing, boxing, tennis and baseball the motor responses must be attuned to the visual data not only with extreme accuracy but with great speed. To enable the boxer to parry a fast blow or the batter to meet a pitched ball the muscular adjustments are controlled by the visual stimuli with really amazing accuracy and speed. Such adjustments might well make use of a short cut of this nature. This function would not be looked on as an usurpation of volitional control but rather as an adjuvant motor pathway serving as a sort of fine adjustment mechanism for volitional acts, and its training might well be the factor which gives the greater speed and accuracy to volitional movements which come with that degree of practice that renders them semi-automatic.

The function of the second level is probably that of the registration and interpretation of objective or pictorial material and may be seen in the ability of aphasic patients, even in certain of those whose greatest loss is in the visual fraction, to make use of vision not only for spatial orientation and avoidance reactions but also in the interpretation by vision alone of the meaning of objects and pictures. This also stands out often in high relief in children suffering from a special obstacle to the acquisition of reading—the condition for which I have offered the term strephosymbolia. Here, in many instances, one sees children with a striking incapacity to understand the printed word but with good visual control of manual movements and often good use of pictorial material.

In one case which I have reported,[1] a boy, aged 16, who was practically unable to read at all, gave evidence of good use of visual material at the lower levels. He was employed in a bakery during his spare time where his task was cutting the proper amount of dough for each loaf, an occupation which requires, as will be seen, an exceedingly accurate visual judgment. He was also skilful with his hands in many things, and in the Stenquist mechanical assembly test obtained a rating equal to that of the highest 1 per cent of unselected army draft recruits. In the Healy pictorial completion test, no. 2, he also made an excellent showing, ranking in the highest 10 per cent of adult normals. Even more striking perhaps was his ability to copy. In his attempts at propositional writing his errors were so great as to produce an entirely unintelligible jargon of neographisms. Writing to dictation was somewhat better, but many bizarre neographisms still appeared. In copying, however, he was able to reproduce in script, in practically letter perfect form, material presented to him in print. When asked for the content of the material which he had just copied he made the illuminating reply "I don't know—I didn't read it." This capacity to copy without reading has often been recorded in visual aphasia, as well as in strephosymbolia, and Allen, in discussing a paper by me,[2] quoted the case of a boy who could copy long lists of addresses on the typewriter and an elaborate payroll on the adding machine and yet was unable to read the addresses he was copying. This capacity to copy without reading would seem to indicate clearly the existence of a motor pathway which permits of responses from the second level without implication of the associative process.

The third level serves, I believe, for the facile interlinking of data brought in by the various sensory pathways. According to current theory, this is the level of residence of the sensory elements of language and it is chiefly here that the associative linkages occur which form the concept. While the motor cortices have not primarily been under discussion here, it may be remarked that the effector cortices comparable to the third level sensory areas are probably those covering the frontal pole in front of the two agranular cortices of the central fields. This zone, I believe, deals largely with the synthesis of the more complex reaction patterns.

Granted then that clinical evidence suggests the existence of pathways whereby the activities of the lower cortical levels may impress themselves on the motor mechanisms, I think that one may draw on two other important theorems in the study of many mental symptoms: (1) that of a

1. Orton, S. T.: "Wordblindness" in School Children, Arch. Neurol. & Psychiat. **14**:581 (Nov.) 1925.

2. Orton, S. T.: The Three Levels of Cortical Elaboration in Relation to Certain Psychiatric Symptoms, Am. J. Psychiat. **8**:647 (Jan.) 1929.

functional degradation or organic disturbance which may be highly selective in its attack within the nervous system, and (2) that of the reappearance of older response patterns by virtue of reduction or loss of control from the higher levels.

SELECTIVITY OF NERVOUS LESIONS

A high degree of selectivity is recognized in many organic diseases involving the lower levels of the central nervous system. Here I need merely cite such processes as poliomyelitis, the familial ataxias and the various lenticular degenerations. Such selectivity is not so widely accepted at the higher levels, but there is some reason to believe that there is a much greater vulnerability of the more recently acquired structures, i.e., those representing the latest evolutionary step in each phylum. Certain it is that here one finds the greatest individual functional variation, and it is probable not only that these higher levels are more subject to check by adverse conditions during the early stages of ontogenetic development, giving rise to various sporadic mental enfeeblements of childhood, but also that they are more subject to deleterious influences affecting their functional integrity after development is complete, resulting in intellectual degradations. The progressive advance of anesthesia seems particularly illuminative of this difference in susceptibility of the various phylogenetic levels. Early in the exhibition of a strong anesthetic there is often a reduction in the critical inhibition, resulting in a rapid flow of rather superficially associated words; then there comes loss of consciousness, but at this stage (excitation period) there is usually a good deal of well integrated but somewhat purposeless motor activity. With the increase of concentration of the anesthetic in the blood this type of pseudovolitional motor activity ceases, but reflex activity of striped muscles can still be demonstrated. This is next submerged, and only the activity of the vegetative centers controlling respiration and circulation remains. This, of course, constitutes about the plane of operative anesthesia, but if in the experimental animal one pushes the anesthetic still further, until death results from failure of respiration, one may still see, on opening the abdomen, orderly caudad peristalsis continuing under the control of the asynaptic net. Another example is seen in the lesser narcosis of acute alcoholic intoxication—first, a volubility and loss of restraint, gradually extending to complete loss of consciousness. It may also be recalled here that Kraepelin held that the chronic alcoholic deteriorations are in essence a degradation of the higher functions comparable to that seen in the acute debauch, but become permanent because of the often repeated toxic insult to the higher centers.

RESURGENCE BY DEFECT

Destructive or deteriorating processes within the central nervous system may exhibit themselves in various ways, but in none, I think, which are more important for recognition than that which I have called resurgence by defect.[3] Certain primitive patterns of reaction which are of normal occurrence in early development are brought under complete subjugation by the higher centers as these mature, but may reappear when the control from above is permanently lost or temporarily lessened. As an example of this may be cited the control of the bowels and bladder. In early infancy, defecation and urination are purely reflex acts; in severe grades of mental defect, in which higher control is always exceedingly scant, this pattern persists, but in the normal child volitional control of these acts is established as he grows older. The original reflex patterns responsible for the early automatic responses have merely been brought under control, however, and are still functionally competent as is shown by their resumption of command in the event of extensive injury to the higher levels of the spinal cord or of extensive destruction of the brain. Another example may be seen in the Babinski reflex. Dorsal flexion of the great toe is the normal response to plantar irritation in the new-born child, but it disappears as volitional motor control over the legs is gained. Kleitmann has shown that the Babinski reflex appears in normal young men in the deep sleep that follows the deliberate deprivation of sleep for long periods. By cinematographic analysis, Rosett [4] has demonstrated the presence of many highly integrated movements and postures, such as supplication, posing, dancing and balancing, which form units of the epileptic convulsion and which are, I believe, to be looked on as related to the resurgent phenomena. This view is reinforced by his observations that in many instances his patients carried out acts in which they had not been trained and which they were incapable of carrying out volitionally. Many other illustrations of resurgence at lower levels might be quoted. Psychopathology has also pointed out the recurrence in many deteriorating conditions of earlier symbolic patterns in the infantile regressions and these, it would seem to me, one may interpret as the psychologic comparates of the submerged neurologic patterns already discussed. This interpretation, moreover, will permit one to view these regressive symptoms as re-emergent because of functional losses at the higher levels, and hence will relieve one from the embarrassment of the etiologic implications of such symptoms encountered when adequate organic etiology is demonstrable.

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3. Orton, S. T.: Neuropathology: Lecture Notes, Arch. Neurol. & Psychiat. **15**:763 (June) 1926.

4. Rosett, Joshua: The Epileptic Seizure, Arch. Neurol. & Psychiat. **21**:731 (April) 1929.

In the acute confusional states of the toxic-infective-exhaustive group a widespread nonselective chromatolysis has come to be accepted as the underlying pathologic alteration. The clinical picture here, with the clouding of consciousness, the defective sensory registration and the consequent amnesia or fragmentary dreamlike recall, is in fairly clear contrast with the typical picture of catatonia in which there is evidence to indicate a much more definitive reduction of the motor functions than of the sensorium. This is rather clearly supported by the occasional registration in memory of words and happenings of the environment during a catatonic stupor, although there has been no reaction thereto to give evidence of their reception at the time. Indeed the whole syndrome of catatonia suggests a reduction of activity of the responding mechanisms at the highest integrative level. Critical thinking, judgment, reason and propositional speech are all reduced out of proportion to the obvious capacity of the person to receive, record and recall experiences. Thus, clinical evidence would point to a differential degradation of the functions of those particular structures which, because of their recent evolutionary development, are felt to be the most vulnerable.

This view permits one to offer a tentative working explanation of many symptoms of catatonia as resurgence phenomena. The catatonic patient shows a reduction in speech varying from a decrease of the recall vocabulary to complete mutism. The more bizarre forms of speech, such as echolalia and stereotypy, have generally been looked on as unexplainable distortions of the speech mechanism. In the development of speech, however, stereotypic reproductions ("lal-period") and mimicry of the speech of others form rather definite phases. That the neurologic mechanisms in use during these stages of development remain intact in the adult may, I think, be assumed. These steps in speech acquisition, while not accurately correlated in time, correspond fairly closely with that period in the development of the child's brain when only the arrival platform is mature. Carrying this over to the catatonic one may tentatively look on echolalia as a direct response from the auditory arrival platform cortex carried forward by the motor "cross over" of the first level, and thus comparable in its mechanization to the mimetic period of the acquisition of speech. Indeed, it would not be necessary to envisage the entrance of such stimuli into the conscious phase of intellectual life, but they may be looked on purely as an automatism due to a failure of the usual critical review by the higher cortices. By this view the word received from without by the catatonic patient would form the pattern of a parrot-like motor response and might do so without leaving a record in memory or without carrying understanding of its meaning. This is in harmony with the feeling of compulsion which many catatonic patients express when they say that they do not know why they responded as they did but just "had to do it." Again, the stereotypy

of phrase may be interpreted as an automatism whereby the first stimulus operates to establish a self-perpetuating responding mechanism in which the self-spoken word, reaching the auditory arrival platform, is again relayed to the motor centers and thus repeats itself. Mimicry of gesture in the child forms a step in the development of motor control by vision comparable to the mimetic phase in speech, and the analogy presented by echopraxia may be interpreted as a resurgence of this older pattern. In the stereotypy of movement a similar factor to that suggested for stereotypy of speech may be envisaged, but with its origin in kinesthesia, viz., an automatic self-perpetuating response engendered by the lower level kinesthetic centers.

As the basis for further critical study and investigation, I think that one may predicate in catatonia a highly selective or differential reduction of functional integrity at the highest level, not only as a direct assumption by virtue of the presence of defect phenomena at this level, as seen in the reductions in speech, in judgment, in reason and in volitional control, but equally by indirection because of the reappearance as resurgent phenomena of older and normally submerged patterns of response. This points the way to a localizable implication of the frontal areas of the brain. Whether one deems the psychopathologic factors demonstrable in certain groups of cases to be etiologically adequate or desires to search further for organic disturbances, this localization is equally apt and permits of a keener understanding of catatonia as a syndrome which may occur in a variety of diseases, some of which are frankly organic in origin. The catatonic episodes which occasionally color the early convalescence from severe toxic conditions might thus be well explained as a more gradual restoration of the third level functions than of those of the older phylogenetic levels. One of my patients who had gone through an extremely severe puerperal toxic confusion during early convalescence gave so typical a catatonic picture that the earlier diagnosis was sharply questioned. She followed, however, a progressive course from the initial delirium through the catatonic episode to complete recovery, and I am inclined to look on that course as an example of the *"per gradum"* recovery of the cerebral levels. Much more intimate clinical studies in such cases, and particularly in those in which convalescence is slow and in which, therefore, the steps are drawn out and appear as fairly clearcut clinical stages, with critical comparison between the presenting symptoms and the developmental steps of childhood, should prove of value in testing this hypothesis.

This incrimination of the frontal cortices in catatonia through interpretation of the clinical syndrome brings up for review the problem of the possible nature of the conditions which might lead to such a selective degeneration of the effector cortices of the third level. The influence of intoxications is obvious here. When severe these are usually not

selective in their effect, but as already reviewed, in progressive narcosis there is evidence of stratification of effect as there seems to be in recovery from the severe deliria. In conditions of accepted toxic origin ultimate recovery is the rule, and one is apt to consider recovery as a diagnostic criterion here. The possibility of permanent damage of selective type occurring in severe toxic infective diseases raises a reasonable doubt as to the justification of the tendency to incorporate patients who do not recover in the schizophrenia group, and to interpret the antecedent puerperal, influenzal or other febrile factors as merely precipitants. In those cases, however, in which such toxic factors are not determinable and which constitute the catatonic form of dementia praecox the absence of readily demonstrable structural changes has left this process on the fence between the functional and organic interpretations. Extensive histologic studies, such as those of Dunlap,[5] have yielded negative results. Alzheimer [6] pointed out, however, that definite alterations are to be observed when death occurs in the acute stages and I [7] reported a case in which such observations in the brain constituted the only demonstrable pathologic conditions in the body. I think it only fair to say here that the technic of today comprehends only a part of the normal structure of the brain, and there is much to be learned before one can entirely exclude organic processes. There is, for example, no really thorough analysis of the normal intracortical nerve pathways and almost no information concerning the extent of synaptic interconnections between the cells of the mantle necessary for normal function; it is therefore entirely within the realm of possibility that, were one prepared to analyze the cortex in cases of catatonic dementia praecox with a really adequate histologic approach, one might find a critical reduction of the synaptic interconnections even in the presence of normal cell counts and normal morphologic pictures. The body of the nerve cell is made up of an intimate mixture of lipoids and proteids. That these mixtures cover a wide range of substances united in various combinations is suggested by the high specialization of various nerve cells —their striking selectivity of response, their selective vulnerability and their differences in inherent rhythm of discharge all indicate differences in chemical composition. This problem offers a promising field for conjecture and experiment which has as yet scarcely been approached.

Histologic changes in the nerve cell give evidence of alterations in the lipoid and proteid structure, but I think it important to bear in mind that integrity of structure does not infer integrity of function. There has been a widespread tendency to confuse the terms structural and

5. Dunlap: Am. J. Psychiat. **3**:3, 1929.

6. Alzheimer, A.: Histologische Arbeiten, Jena, Gustav Fischer, 1910, vol. 3, pt. 3.

7. Orton, S. T.: A Study of the Brain in a Case of Catatonic Hirntod, Am. J. Insan. **69**:669, 1913.

organic here and to assume that a process which leaves no microscopic trail must therefore be looked on as functional in origin. Microscopically demonstrable changes in the cells (i.e., structural changes) represent only a part of the organic possibilities which may lead to disturbance of function, and in addition to a normal structure of the proteids and lipoids which constitute the cell body and a supply of these materials to replace wear, there must also be a constant and adequate supply of oxygen and of blood sugar and, I believe, an extremely accurate balance between the monovalent and divalent salts in the pericellular fluids. I have elsewhere reported a series of experiments in which death in convulsions was induced and yet in which there was no histologic evidence of change in the nerve cells. Sudden cerebral anemia produced by ligation of both carotid arteries in the dog, reduction in blood sugar by toxic doses of insulin in the dog and rabbit, intravenous administration of oxalic acid in the rabbit and tetany parathyropriva in the dog all resulted in death in acute convulsions, but in none was consistent damage to the nerve cell revealed by even the most careful histologic study with a wide choice of technical methods.

To what extent such organic but nonstructural disturbances might be established on a sufficiently permanent basis to account for chronic processes such as dementia praecox one cannot yet answer, but their consideration opens anew the interest in vascular inadequacies, chronic vasomotor disturbances, disorders of the internal secretory apparatus, etc.

Modern studies in the familial occurrence of dementia praecox also bring up for review certain possibilities. In some organic degenerative diseases of familial occurrence one recognizes a cacogenic influence which is selective in its type. Thus, the degenerations of the type seen in Friedreich's ataxia and familial spastic paraplegia imply an inherent weakness not of the nerve tissues as a whole but of the gangliospinal and corticospinal groups, respectively. Obviously, such a hereditary weakness might operate to render such limited systems either more susceptible to toxic degenerations or more apt to suffer from precocious selective senile changes.[8] This latter vision of a hereditary limitation of the natural life span of a selected group of nerve cells can scarcely be of interest in a process in which cell disintegration does not come to the fore, but the former, that of a cacogenetic susceptibility to certain toxic or other deleterious conditions on the part of a portion of the nervous system, especially when considered in relation to the greater vulnerability of the cortices of recent evolution, may well be a factor of importance.

The views here given are not intended as by any means an exhaustive review of the possibilities of catatonia, but merely to review certain patent opportunities for clinical study and laboratory investigation.

8. Orton, S. T.: The Pathology of the Hereditary and Familial Nervous Diseases, Arch. Neurol. & Phychiat. **13**:96 (Jan.) 1929.

ABSTRACT OF DISCUSSION

DR. F. TILNEY, New York: As might be expected of one whose interpretative inclinations are strongly organic rather than psychogenetic, Dr. Orton's three levels of the cortex platform appeal to me strongly, particularly as applied in the solution of psychiatric problems.

Orton has pointed out the structural foundations of what he chooses to call three platforms, and in his extensive work on the cortex he has given most of his attention to the visual area, in which he recognizes the striatal area as the first level, the parastriatal area as the second level and the posterior-association region as the third level.

He has also shown that these three levels or platforms are susceptible of functional separation, that is to say, that there may occur between these platforms a detachment so that one or the other may be separated in its action. This functional separation brings up the important point that he has made of seeing exactly what functions he attributes to each level.

The first level he calls the platform for awareness of the origin of sensation, that is, for current sensation without mnemonic recall. The second level is higher than that, and he regards it as objective recognition, or recognition of objects with mnemonic recall, with memory associations rather indefinite and diffuse, but, nevertheless, definite recall. The third level adds the conceptual or symbolic elaborations and associations, chiefly those of language.

On each one of these three levels, he predicates a type of behavior and interprets this behavior in the light of receiving mechanisms of each separate level of the cortex.

At the present time, I have under my care at the Neurological Institute a patient whose case illustrates exactly these levels of reaction and behavior which Dr. Orton has called to our attention. I should like, in the analysis of this case, to apply Dr. Orton's three levels or platforms of the cortex. The patient is a woman 20 years of age. She came to me suffering from a peculiar cycle of symptoms and with a variety of diagnoses. When she arrived at the institute, she was affable, agreeable and altogether delightful in her manner. She told me that she had come a long distance from the country. She told me about the trip and that she hoped she would get cured of this disease which troubled her.

Two days later she had a series of eight convulsions and immediately thereafter began to show marked change in her behavioral reactions. She became markedly retarded. When asked a question she would reply, at first, after a lapse of ten seconds, then twenty, and finally her replies were merely repetitions of the questions asked her, and at length, only single words continually repeated from the question.

In the course of two or three days, she lapsed into complete mutism and was thoroughly catatonic. She remained in this state for three days and then a rather sudden and sharp change took place in her. She became extremely antagonistic and abusive, especially to me and to her father, and also to the nurse. She ordered us out of the room and away from her and in every way showed strong anatagonism toward us. I am certain that she did not recognize any of us, although she apparently had some association in her mind that we represented an authority over her and were thus antagonistic to her.

We treated her for several days in a continuous bath, and at the end of a week she emerged from this condition, became affable again, was pleasant to us, recognized me, called me by name, and, strangely enough, had some dim recollection of the episode through which she had passed.

This all occurred in a period of about two weeks. Her father, a clergyman, and an intelligent man, said that his daughter had passed through this same cycle several times before, only heretofore it had been over a much longer period of time, six or seven months, and that she had compressed within the limit of two weeks the whole episode for our observation.

I was much perplexed about making a diagnosis under the circumstances but an ensemble of her symptoms has cleared that matter. She has a paralysis, a flaccid paralysis of the left leg, a slight paralysis on the left side of the face, an external strabismus in the left eye, and she had at one time a marked degree of diplopia. These facts taken in conjunction with her convulsions and also the statement that her trouble, which was considered an acute poliomyelitis, began several years ago finally led me to believe that the diagnosis was encephalomyelitis disseminata.

Dr. Orton has shown that these three levels on which such marked behavior disturbances may occur can be dissociated under the influence of certain toxins. He has mentioned particularly the effects of anesthesia and of alcohol. It is probable also that many other toxins may act in this way, and it seems conceivable that the toxins consequent on vaccinia, or even the virus of such conditions, following measles, or scarlet fever, or many other infectious conditions may produce this type of dissociation.

In any event, while we may be unwilling to ascribe these changes to definite organic disturbance of the brain—organic in the sense that it has produced a permanent disintegration—there are surely, short of this, many biochemical changes which in extent amount to definite organic disorders.

I feel that in the approach to psychiatric problems of any kind, a variety of methods of interpretation must be employed, but this neurologic concept or, as I choose to consider it, organic concept, in the interpretation of psychiatric conditions is one, particularly as applied to catatonia, which must have thorough testing, both experimentally and clinically.

Dr. Adolf Meyer, Baltimore: In consideration of the additional illumination of the point of view by Dr. Tilney, I cannot help but ask myself whether I would spontaneously, if I were not requested to do so, speak in the discussion of this interesting account of the organicist's musings and considerations in connection with the possible application of the neurologic issues to the problem of psychoses and particularly catatonia.

Somehow, as a pronounced pluralist, one who gives due consideration and keenly interested consideration to the variety of accessible and workable aspects that come into one's field in the consideration of psychoses, I naturally turn gladly, in such a discussion as Dr. Orton's, to that field which he particularly singles out. But I am accustomed to distinguish neurologizing tautologies from specifically neurologic problems and sets of facts. I speak of neurologizing tautologies when data which we know only from other angles or from inferences are presented in hypothetic neurologic terminology.

I like to give consideration to the neurologic sets of facts and the hypotheses that have been touched on when we can use actual neurologic data and processes; particularly when we come to such definite localizing distinctions, I like to look for those disturbances that we actually know as localized processes and their consequences.

I am, therefore, taken back to some extent to such material as I discussed in the Harvey lecture of 1910, when I tried to use the concept of integrational units in connection with the cortical zones underlying Dr. Orton's presentation, when in the visual apparatus I recognized an hemianopia issue, then the mind-blindness

issue, then the associative issues, associative partly to language and partly to motility, the perception of depth and things of that sort; in a similar way integrational units in the auditory sphere and also in the sensorimotor sphere. There I have the feeling that I am in the presence of definite facts with an accounting of what functional processes I observe and have a chance to correlate, and also an opportunity and duty to test my correlations with the autopsy investigation and the study of the nervous system.

On a matter of that sort, I am exceedingly keen; but, on the whole, I suppose I have, just on account of that keenness, a certain reserve about thinking in anatomic terms. When the facts are not available and not provocative or productive, I may have to go rather in the direction of an appeal to other complexes of integration that deal with the issues in question.

When we come to such a term as catatonia, I cannot help but feel that it was, in a way, one of the saddest things that happened to Kahlbaum when he had the impression that the disease entity of paresis, with its paralytic defect phenomena, could be paralleled by a similar entity or psychosis with motor processes of a positive type, the type of overactivities or unusual activities, activities that Dr. Orton classifies largely from an angle that would be excellent if we dealt with something accessible to the surgeon and operative therapy. Kahlbaum himself did not think of those things in just those terms of localization, but the idea was to contrast a negative or paralytic process and a positive motor process and to develop a disease entity out of it.

With all the experience that I have had with catatonic symptom-complexes, or, I might say, catatonic manifestations, I have to try to get at a cardinal something that holds the facts together, according to the case in which it occurs.

Dr. Orton has particularly referred to mutism as a reduction of speech. One condition in which stupor and mutism are produced by focal disease we are familiar with particularly, viz., in cases of abscess of the temporal lobe (i. e., deeper and nutritional rather than destructive involvement of the auditory cortex), in which one often gets preeminently the mutism, the abolition of the initiative, and also, probably, of the more complex uses of the functioning.

It is also interesting to note that Southard in his enthusiastic attempt to use the palpation method for the determination of lesions came to focus his attention largely on the postcentral regions to localize catatonia, whereas it is the frontal lobe that Dr. Orton particularly referred to, and as many of us would have expected even in 1910, in view of the studies of Kleist.

Just where do we stand on this question? Unfortunately, lesions of the frontal lobe may give fragments of catatonia-like components but not catatonia. After all, we deal to a certain extent with an attempt at harmonizing fragmentary observations in terms of what we would like to represent in localizing terms, in terms of organic neurology. Guiraud and others (including Kleist in his later work) have especially pointed to the striatal and extrapyramidal conditions. There is no part of the nervous system that does not form here and there a contributory factor to the pyramid of integrations that Dr. Orton has outlined.

What I want to emphasize is that I like to pay attention to what is organic, what is neurologically localizable and can be thought of and tested in that way. But I should like to ask what one should teach the beginners and the research workers when the study of catatonia is approached. What should one urge them to single out and work on? I should have liked to hear from Dr. Orton something not so much as to the things that followed, but the antecedents of the person who went through a delirium and such a characteristic solution of the whole attack in terms of a catatonic reaction. That would have been a supplementary

accounting. I would accept the accounting for the delirium as evident—a toxic process. But, then, why that particular solution? I would advise a student and a researcher working with me to be sure to make a number of spheres of study; to study, above all things, the things which he has to handle while they are there alive in the patient, which have to be taken up more especially in terms of function—that is naturally also organic—but as functions of the organic, and I have to express these functions of the organic in terms of actual happenings and functions, not in terms of hypothetic "cross-overs."

Therefore, I should say that the student has to learn to focus his attention, as far as work is concerned, on the functional mechanisms that we can test, that we have to apply our therapeutic efforts to, and that we have to test from a theoretical point of view as well.

Then by all means, particularly if one has the opportunity for autopsy and to plan anatomic investigation, one again has to think of what to do, what had best be worked out anatomically. That is the time particularly for the thinking and planning in terms of "postmortem symptomatology," the anatomic study of that which remains over in the corpse, and I might then be enthusiastic over guidance that Dr. Orton might be able to give us.

I should therefore express myself in this way: that I am delighted to hear that the neuro-organicist comes with his musings concerning the organic facts back of catatonia to things which I specially enjoy investigating in the focal lesions, and that there is promise of harmony in nature and in the world. Then comes the task of bringing the functionalization clearly before us. When we come to teaching therapeutically promising conceptions and to the actual research work, a physician would do well to recognize several integration complexes and integration fields which he must not feel under obligation to confuse with one another or to substitute for one another too easily.

The problem of catatonia will for some time be more adequately connected with those things in which we learn to know functionally when catalepsy, echolalia and similar conditions occur with experimentally accessible regularity.

I get a great deal out of harmonization with those automatisms that I know from the hypnotic submission reactions; I there get vivid and immediate correlation with what I see in the history, the personality and the entire development and what I am able to use experimentally and to try out, in a way, for comparisons and for kinship of the conditions under which the similar developments occur. I do not make an absolute identification, naturally. When the problem comes of trying to find out whether there are toxins, whether there are fever processes, whether there are, perhaps, organic processes (because we can have traumatic pseudocatatonia), I am keen to look for pertinent evidence. I also would see to it that my organic reasoning could be mobilized in proper time. But I also keep my mind open to what still appears evident, viz., the sets of developments which we know as not primarily focally produced automatims.

Dr. Foster Kennedy, New York: Dr. Meyer speaks of Dr. Orton as an organicist. I wonder if it is possible to differentiate neurologists and psychiatrists so completely and discretely by their records and their interest.

Surely it is the desire of all to learn, if possible, the union between mind and body, and we neurologists, if I may accept Dr. Meyer's differentiation, feel indeed that we are psychiatrists with insight into our own ignorance. We know that we have no pathology of mind and we are eager for it. I feel, on hearing Dr. Orton's paper, that here is a definite effort to unite behavior with mechanism. In our work we are constantly seeing evidences that with disintegration and dissociation of

personality, there is clear evidence of splitting of organic function of mechanism. It is so in such a simple affair as in an onslaught against an optic nerve, in which the phylogenetic youth of the macular bundle allows it to degenerate without any difficulty being spread into the other fibers.

This also is true in all senile states, and I should like to cite in epitome the case of a soldier under my care who was injured by a heavy shell flash in a dugout. Several of the men in the dugout were killed. He was taken out for dead. He was unwounded but unconscious and remained so for two weeks. He then recovered consciousness, but was completely catatonic. He then recovered from this catatonia but remained mute. He developed a pure striatal lesion, having the evidences of a pure striatal subject, with decerebrate rigidity; the last stage of all was abnormality of the spinal cord. He passed through the whole gamut of recovery from the cortex to the cauda over a period of four months.

Even hysteria completely imitates in many of its manifestations a striatal picture, so that one of the difficulties of diagnosis is to distinguish between organic conditions of the midbrain due to infection, such as encephalitis, and hysterical phenomena.

We find encephalitis producing neuroses, about the diagnosis of which we would not have the slightest hesitation if there had not been concomitant organic conditions; we find encephalitis producing phenomena identical with dementia praecox—they are indistinguishable. I can bring to mind a couple of cases in which the condition was diagnosed as dementia praecox by members of this body and maintained as such in excellent hospitals for the insane, and in which the patients were later proved to be suffering from encephalitis.

Dr. Orton has in his final words refused to accept the assumption that, because after but fifty years' experience in examinations of the brain by histologic methods we are unable to find changes in the brain, there can be, therefore, no structural changes in the brain. That is a presumptuousness that no sane man ought to assume.

DR. ORTON: There is little for me to say in closing the discussion.

Dr. Tilney's case was particularly interesting and I think illustrates the type of thing that I, myself, have been interested in, in this fractionation which can be seen if this vision of cerebral function is held.

With regard to Dr. Meyer's statements, I have absolutely no fault to find with the use of everything in pragmatic or clinical methods, or for teaching, and I think, with him, that the functional study should be followed and should be used in teaching, but the problem is deeper than that. The problem is, really, what is going to be the background of our research approach? The teaching, after all, is a pragmatic affair, whereas the research has a good deal deeper basis, is a good deal more fundamental, and if in our fundamental or basic thinking we are ready to accept a functional interpretation, we are led astray from what must be the ultimate goal, not of our generation, but of some future day, when function can actually be related to structure. I believe that the human brain is the result of a long period of evolution and that absolutely all of the functions of the brain are dependent on activities within its structures, and that is the rock on which the organicist stands.

I do not mean to say that we are prepared in any way to meet that situation yet, but I think that in our ultimate vision we must bear in mind that approach to all research problems, regardless of how pragmatic points of current treatment or current teaching may lead us astray from it, it must form the backbone of our research program.

STUDIES ON CEREBROSPINAL CIRCULATION BY A NEW METHOD*

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One of the many questions concerning cerebrospinal fluid that has never been definitely determined is whether there is a true circulation in it. In order to state that a fluid has a true circulation, certain criteria must be fulfilled; first, and most important, there must be a constant movement of the fluid in a given direction and along certain pathways; second, particles injected into this current must be carried along in this one constant direction by the fluid.

Numerous investigators have studied the subject. All these studies may be grouped under one of the following headings:

(1) The method and place of production of cerebrospinal fluid
(2) The pathways of the fluid
(3) The pulsation of cerebrospinal fluid
(4) The pressure of cerebrospinal fluid, particularly in relation to arterial and venous intracranial pressure
(5) The place and method of absorption of the fluid

REVIEW OF THE LITERATURE

The question of the place and method of production has been investigated by Dandy and Blackfan, Weed, Becht and Gunnar, Solomon, Thompson and Pfeiffer, Dixon and Halliburton, Cushing, Kubie, and Howe. The weight of evidence of these and other investigators tends to show, as concluded by Weed (1914): "The cerebrospinal fluid appears to be derived from two sources: (*a*) the choroid plexuses in the cerebral ventricles, (*b*) the perivascular systems of the nervous tissues."

The pathways of the cerebrospinal fluid have been the subject of the greatest number of investigations, Magendie and Quincke being the

* Submitted for publication, May 1, 1929.

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* Read at the Fifty-Fifth Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 28, 1929.

pioneers in this work. Key and Retzius, and Locke and Naffziger have done the most striking anatomic work, while Hill, Weed, Dixon and Halliburton, Dandy and Blackfan, Stepheanu-Harbotsky, Solomon, Thompson and Pfeiffer, Strecker, Lewis L. Redford, Kramer, and Ricquier and Ferrara have pursued physiologic methods in studying these pathways. These workers have shown that the pathway of the cerebrospinal fluid extends from the cerebral ventricles through the foramen of Monro to the third ventricle, then through the aqueduct of Sylvius to the fourth ventricle; from there through the foramen of Magendie and the lateral clefts of Luschka to the subarachnoid spaces at the base of the brain, beneath the cerebellar tentorium, and the cisterns of the pons, medulla and cerebellum. From here the greater portion passes over the surface of the midbrain through the incisura tentorii and finally to the large venous sinuses, and the lymphatics along the cranial nerves. The lesser portion descends down the subarachnoid spaces of the spinal cord. These studies have shown the possible pathways but they have not thrown light on the actual movement of the fluid.

Movements of the spinal fluid have been studied as early as 1700 by Pacchionis and by Donner in 1850, and more recently by Hill, Becher, Knoll, Salath, Duret, and Howe. The only movement observed has been a pulsation synchronous with respiration and cardiac systole. None of the investigators has shown that the spinal fluid has a definite movement in a given direction, such as would occur in any circulatory system. In fact, all of the evidence presented has shown that no definite movement in a given direction, such as would occur in a true circulation, has been observed.

Hill, Weed, Dandy and Blackfan, Becht and Gunnar, Kubie, and Howe have done the most important work on the cerebrospinal fluid pressure and its relation to intracranial arterial and venous pressure. This is well summarized by Howe (1928) in his conclusions:

> The normal pressure is maintained by the secretion of sufficient cerebrospinal fluid to distend the spinal dura moderately. The intracranial pressure is roughly equal to the venous pressure and, within limits, variations in the pressure in either the cerebrospinal fluid or the venous blood are accompanied by similar directional pressure changes in the other fluid.

The method and place of absorption of the cerebrospinal fluid have been much discussed, and various theories have been advanced. Cathelin, Papillian and Jippa claim it is absorbed through the lymphatics, while Weed, Mort, Dandy and Blackfan, Howe, Hill and others maintain it is absorbed into the venous system. But while Weed believes it passes into the venous system through the pacchionian bodies, the others believe it passes directly into the blood vessels.

Dandy and Blackfan believe that the fluid is absorbed along the entire cerebral nervous system; Weed concurs in this idea, but believes that the cranial portion is more efficient and that there is an accessory drainage into the lymphatic system.

Cushing, in his "Cameron Lectures," first spoke of a third circulation, and in support of this idea quoted the work of Lewis L. Reford who injected india ink into the spinal subarachnoid space of pig embryos and found that it traveled cephalad to the ventricle and around the base of the brain over the hemispheres. Hill, Schwalbe and others were able to inject the vertebral canal by making subdural injections over the cerebral cortex. All the observers quoted by Hill, however, agreed that injections through the atlanto-occipital ligament spread both upward to the ventricles and downward into the canal.

Dandy and Blackfan showed that phenolsulphonphthalein injected into the lumbar region was recovered from the ventricles, and when injected into the ventricles could be found in the lumbar region.

Dixon and Halliburton (1915-1916) found that drugs and dyes, such as methylene blue (methylthionine chloride, U. S. P.), carmine and potassium ferrocyanide, injected into the occipito-atlantoid ligament passed into the ventricles and over the brain, but that they also passed downward into the vertebral canal, diffusion being most rapid in the subcerebellar region and extremely slow in the lower spinal tract.

Weed (1917-1918), in his description of the course of the dye injected into the ventricle, described the course as being from the ventricles downward to the subcerebellar subarachnoid spaces and also diffusing downward into the spinal subarachnoid spaces. All this evidence is conflicting; it shows that substances injected into the lumbar region travel upward toward the ventricles, and that substances injected into the ventricles pass downward to the lumbar region.

Howe (1928) raised the question whether or not an active circulation exists, stating:

> There is much to support the view that under normal conditions the exchange of cerebrospinal fluid is slow. The difference in the contents of the fluid when obtained simultaneously from different loci would evidence a sluggish circulation which would not be the case were the fluid undergoing rapid secretion and absorption. The slow diffusion and unequal distribution of foreign particles such as India ink when injected into the subarachnoid spaces point to the same conclusion. It is difficult to see how a rapid exchange of this fluid could serve any useful purpose, and as its main function seems to be mechanical; stability, with but slow circulation or exchange, would seem more physiologically advantageous.

Weed (1923) stated: "In the process of spread of the fluid the normal currents of the cerebrospinal fluid and the *physical phenomenon of diffusion* between the normal fluid and the foreign solution play the important parts."

The evidence is overwhelming that cerebrospinal fluid originates both from the choroid plexus and from the perivascular spaces of the cerebrospinal axis, and that it is absorbed both in the venous sinuses and in the spinal subarachnoid space.

Though the fluid is both produced and absorbed in various regions, there is no good reason to postulate that a circulation exists any more than that there is a circulation in the large serous cavities where fluid is also produced and absorbed.

Various methods to determine circulation have been used. These may be grouped under six headings:

(1) The recording of oscillations, used by Becher and Knoll.
(2) The puncture method, used by Frazier and Peet.
(3) Making casts of the pathways, used by Key and Retzius and by Locke and Naffziger.
(4) The physiologic action of drugs following their injection, such as phenolsulphonphthalein, tetrahydro-B-naphthylamin hydrochloride, epinephrine and pituitary extract, used by Strecker, Ricquier and Ferrara, Dandy and Blackfan, Dixon and Halliburton, and others.
(5) The injection of true solutions with subsequent precipitation, used by Weed, Dixon and Halliburton.
(6) The injection of macroscopic particles in suspension and their subsequent observation either by withdrawal at another place by puncture or by their observation in situ post mortem, as done by Buia, Babes, Solomon, Thompson and Pfeiffer, Goldman, Kramer, Quincke, Cushing, Weed, Hill, and Dixon and Halliburton.

The recording of oscillations of the fluid does not indicate that an ultimate directional movement of the fluid does or does not take place since a noncirculating fluid may oscillate and a circulating fluid such as the blood, may pulsate.

The puncture method is obviously false in that pressure is reduced in the system and an artificial circulation is established; an abnormally rapid production of the fluid may occur, such as occurs in ascites where the normal pressure relations between the vascular system and the fluid in the cavity have been altered.

The injection of viscous fluids under abnormal pressures displacing the spinal fluid mechanically, as was done by Key and Retzius and by Locke and Naffziger, does not show that the course taken by the injected material is the directional course of the cerebrospinal fluid under normal conditions.

The physiologic action of drugs following their injection into the cerebrospinal fluid indicates only that they have been absorbed from the spinal fluid into the blood stream at any point in the axis, since it has been shown that absorption may take place at any point.

The injection of true solutions or of foreign particles in suspension and their subsequent observation in situ post mortem does not show

that they were necessarily carried by a circulation unless the direction of their transportation was constant. This transportation of the particles or true solutions in a constant direction has not occurred; since, following the injection of true solutions or foreign particles in suspension, they have been recovered or observed in situ post mortem in the lumbar region, when injected into the ventricles, and have been recovered from the ventricles when injected into the lumbar region.

An analysis of the previous work pointed rather to the idea advanced by Dixon and Halliburton, that diffusion played a great rôle in the spread of substances in the cerebrospinal fluid. If this were true, then the rate of spread would be no more rapid in the living animal than in cerebrospinal fluid placed in a sealed tube. On the other hand, if there were a true circulation, the rate of spread would be equal to the diffusion plus the rate of flow and would be more rapid in the animal in vivo than in a tube.

METHOD

The method of study to clear up this point, therefore, was to inject a dye, trypan-blue, into the closed subarachnoid space and observe its movements.

Trypan-blue in isotonic saline solution or cerebrospinal fluid is a colloid suspension. In order to observe the spread of the dye without markedly altering normal conditions, that portion of the dura to be studied was freely exposed, and then with a fine hypodermic needle the dye was injected at one point and its spread observed. The point of injection was immediately sealed with a piece of muscle, so that the subarachnoid system remained closed during the entire experiment.

All dogs were operated on under ether anesthesia. Forty-one dogs were operated on; of these, nine were discarded because of leaks at the site of injection. Two hundred milligrams of trypan-blue, in from 1 to 1.5 cc. of isotonic saline solution or cerebrospinal fluid, was used for injection purposes. The specific gravity of the fluid before addition of the dye was 1.007; with the dye in colloidal suspension it was 1.025. In order to disturb pressure conditions as little as possible, 0.5 cc. of the colloidal suspension was put into a syringe; then 0.5 cc. of cerebrospinal fluid was drawn up into the syringe. This diluted the solution 100 per cent, and 0.5 cc. of this mixture was reinjected. The procedure took but a few seconds, not long enough for an increased production of cerebrospinal fluid to take place as a result of the momentary slight lowering of the pressure. Following the injection the total volume of the fluid in the central nervous system was unchanged, as was also the intradural pressure.

Immediately following the injection, a definite margin of dye could be seen through the dura, pulsating cephalad and caudad. The extent of pulsation varied from 0.5 to 1 cm., depending on the depth of respiration. The point taken for the initial measurement was that point at which the margin of the dye was farthest from the point of injection, during the period of its oscillation. The point was marked immediately after the mass had been injected. At various intervals of time, the dye margin was measured as it spread from this initial point up the cord. These measurements were recorded as shown in table 1.

As will be seen in table 1, the average diffusion rate for a given interval of time was determined by dividing the number of centimeters diffused in that interval of time, by the time interval in minutes. For example, it will be seen that in the first four minutes following the injection, the dye margin had diffused 1 cm. The average rate of diffusion for this time interval of four minutes was then 2.5 mm. per minute. After a variable interval of time, the diffusion rate became so low that two measurements, not less than four minutes apart, were the same; then no further measurements were taken. This gave a diffusion rate for the margin of the dye mass of zero for that time interval. It cannot be emphasized too strongly that this does not indicate that no further spread of dye particles took place; for such spread does take place and will continue to do so, according to the physical laws of diffusion, until an equal distribution of particles throughout the entire mass of fluid has taken place, provided the effect of gravity

TABLE 1.—*Series 1; Experiment 6; Diffusion of Dye after Injection*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:39 p. m., injection.....	...	..	...	.....
3:43 p. m.....	1.0	4	1.0	2.500
3:51 p. m.....	2.0	8	1.0	1.250
3:56 p. m.....	2.5	5	0.5	1.000
4:08 p. m.....	2.8	12	0.3	0.250
4:13 p. m.....	2.9	5	0.1	0.200
4:17 p. m.....	2.0	4	0.0	0.000
Total time.....	...	38		

can be eliminated. It was observed also that as diffusion progressed, the dye margin became less distinct until no definite margin could be seen, and only an area of blue varying in intensity from the deep blue near the point of injection to the clear white of the uncolored spinal fluid above was seen. This confirms the observations of Goldman. As it was obviously impossible to make accurate measurements when the dye margin had become indistinct, we stopped taking measurements when no change was noted in four minutes. Invariably at this time the dye margin had become indistinct, and further measurements were impossible. At the end of ten hours, however, it is still possible to determine that the dye has not diffused throughout the cerebrospinal fluid. Three experiments, one in series 1, one in series 4 and one in series 6, were observed for ten hours, and at the end of that time in none of them had the dye diffused throughout the entire cerebrospinal space. In the first of these, when dye was injected at the third lumbar vertebra, at the end of ten hours it had reached the level of the first cervical, and slight diffusion was noted into the basal cisterna and under the pons. In the second, when injected into the ventricle, it was found at the level of the ninth dorsal vertebra and had spread

over both cerebral hemispheres and the vermis. In the third, when injected into the subarachnoid space in the parietal region, it was found at the level of the sixth cervical vertebra, over both cerebral cortices, the vermis and, most important of all, in both lateral ventricles. The details of these experiments are recorded in protocols 12, 29 and 33.

The following series of experiments were carried out.

1. With the dura exposed from the first dorsal to the first sacral, dye was injected at the lower level of the third lumbar. The number of experiments was twelve.

2. The same exposure as in series 1 was used; the blood pressure and respiratory rate were recorded. Four experiments were carried out.

3. With the cerebral and cerebellar dura exposed and the cervical and dorsal dura exposed, dye was injected at the level of the first dorsal segment. One experiment was done.

4. With the cerebral and cerebellar dura exposed and the cervical and dorsal dura exposed, dye was injected into one ventricle. Twelve experiments were carried out. Series 4 (*a*) was first done with the head higher than the spinal cord. Six experiments were done. Series 4 (*b*) was then repeated with the ventricles lower than the level of the spinal canal, five experiments being done. One experiment was carried on for ten hours, and in this one the ventricles were level with the spinal canal.

5. A glass tube, 5 mm. in diameter and 30 cm. long, was sealed at one end and completely filled with cerebrospinal fluid. The other end was covered with a rubber diaphragm. With the tube in the horizontal position, tested by a spirit level, dye was injected through the diaphragm by the same technic used on the dogs. The same experiment was carried out with the tube at an angle of 45 degrees and the diaphragm at the bottom, and another with the diaphragm at the top.

6. Dye was injected into the subarachnoid space in the parietal region.

In eleven dogs used in series 1, the curve of the rate of diffusion rapidly slowed down, so that at the end of from forty to fifty minutes no appreciable rate of diffusion could be determined. The rate of diffusion was slightly slower than that noted in series 5 when dye was injected through the diaphragm into the glass tube held in a horizontal position. The dye diffused in the tube first at the rate of 10 mm. per minute; at the end of thirty-five minutes, at 0.5 mm. per minute. In the tube only diffusion was a factor, since gravity was excluded by having the tube horizontal.

At the end of twelve hours, the entire tube was uniformly colored.

This seems evidence that dye particles may spread from one point in the cerebrospinal fluid to another without the aid of any circulation.

Series 1 showed that particles may spread in cerebrospinal fluid from an initial point of injection without the aid of any circulation, and the rate of diffusion both in vitro and in vivo shows the same type of curve, the rate decreasing rapidly at first until the curve flattens out, so that the rate can no longer be measured. In no case was the rate of diffusion in vivo maintained at anything like a uniform rate as would be expected if a current existed in the cerebrospinal fluid, and in no case was it as rapid as in the tube experiment.

In the experiments the dye was injected into the lumbar region, and its rate of diffusion up the cord was measured. This point of injection was taken in order to have as long a portion of the subarachnoid space as possible for observation. It was noted in the course of the experiment that the dye also spread caudad from the point of injection to the conus and stopped, as no farther spread in that direction was possible. We have stated that a true circulation exists when

TABLE 2.—*Experiment 30. Diffusion of Dye in a Horizontal Glass Tube*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
5:13 p. m., injection.....	...	..	...	.....
5:14 p. m.....	1.0	1	1.0	10.000
5:18 p. m.....	2.0	4	1.0	2.500
5:28 p. m.....	3.0	10	1.0	1.000
5:48 p. m.....	4.0	20	1.0	0.500
Total time.....	...	35		

particles are transported constantly in one direction. If the dye is injected in the same manner into the femoral artery, it is immediately carried downward by the rapid circulation, and no dye appears above the point of injection. It is conceivable that a circulation might exist that was so sluggish that at first the diffusion rate of the particles was more rapid than the circulation, and for this reason dye particles would be found both above and below the point of injection. When the diffusion rate has slowed up, however, one would expect that the dye would be carried farther away from the point of injection in the direction of the current than it would in the other direction, since the current would tend to carry the particles back toward the point of injection from the point to which they had been carried against the current by a very rapid initial diffusion rate. For instance, if, as claimed by Cushing, there is a circulation upward in the cord, then dye might spread both upward and downward from the point of injection; but it would spread more rapidly upward, since the rate of spread upward would be equal to the sum of the diffusion rate plus the circulation rate, while the rate of spread downward would be equal to the diffusion rate minus the circulation rate. In order to have any spread

downward, it would of course be necessary to assume that the current was so sluggish that the initial diffusion rate would be greater than the rate of the current. Therefore, it is obvious that even the slightest current would spread the dye farther upward than it would downward, if the current were upward as claimed by Cushing.

Series 1, therefore, seems to prove definitely that there is no evidence for believing that there is an upward current in the cerebrospinal fluid.

Since a number of observers (Schwalbe, Lunde, and more recently Dixon and Halliburton) noted that foreign particles injected through the atlanto-occipital ligament traveled both upward and downward, their observations might be explained by a circulation so slow that the initial diffusion rate was great enough to spread the dye in both directions,

TABLE 3.—*Series 3. Experiment 17. Diffusion Measured in Both Directions from Point of Injection*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute	Remarks
		Diffusion Up the Cord			
3:47 p. m., injection ...		..	...	.....	At end of injection at first
3:50 p. m.....	2.7	3	2.7	9.000	dorsal dye margin was at
3:55 p. m.....	4.0	5	1.3	2.600	fifth cervical
4:00 p. m.....	4.1	5	0.1	0.200	
		Diffusion Down the Cord			
3:47 p. m., injection ...		..	...	.....	At end of injection at first
3:50 p. m.....	2.8	3	2.8	9.333	dorsal dye margin was at
3:55 p. m.....	4.1	5	1.3	2.600	fourth dorsal
4:00 p. m.....	4.2	5	0.1	0.200	

but none of these observers noted accurately the comparative spread upward and downward.

In order to answer this vital question, we performed the following experiment:

A laminectomy from the first cervical to the tenth dorsal was performed. The dog's head was lowered so that the cord was as nearly horizontal as possible. Dye was injected in the same manner as described before, but at the level of the first dorsal vertebra. At the end of the injection, the dye margin had reached the fifth cervical upward and the fourth dorsal downward. Measurements of the rate of diffusion, both upward and downward, were then taken. As shown by table 3, experiment 17, series 3, the rate of spread of the dye up and down the cord was identical. This seems to show rather conclusively that there is no movement of the cerebrospinal fluid in a constant direction, either up or down the cord.

To determine if pressure changes or oscillatory movements might be factors responsible for the spread of substances injected into the cerebrospinal fluid, four experiments were done in which pulse rate and respiration were recorded. Protocols 13, 14, 15 and 16 reveal no

correlation between pulse and respiration and diffusion rates. The variations in pulse and respiration rates were obtained by varying the depth of the anesthesia.

The effect of gravity was considered another possible factor in the spread of substances injected into the cerebrospinal fluid. Weed and others had noted in postmortem examinations with the organs in situ that particles injected into the cisterna were found to be densest around the base of the brain. Such an observation might be explained by the effect of gravity. If gravity were a factor, one would expect that the rate of diffusion downward would be equal to the sum of the normal diffusion rate and the effect of gravity, and would therefore be more rapid than if gravity played no part. Secondly, the diffusion

TABLE 4.—*Effect of Gravity in Vitro. Tube at 45 Degree Angle; 0.5 cc. of Trypan-Blue Injected at Top: Diffusion Down*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:53 p. m., injection.....	..	..	..	.....
3:55 p. m.....	6	2	6	30:000
3:56 p. m.....	10	1	4	40:000
3:57 p. m.....	13	1	3	30:000
3:58 p. m.....	15	1	2	20:000

TABLE 5.—*Tube at 45 Degree Angle, 0.5 cc. of Trypan-Blue Injected at Bottom: Diffusion Up*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
4:25 p. m., injection.....	...	..	...	.....
4:28 p. m.....	0.5	3	0.5	1.666
4:34 p. m.....	1.0	4	0.5	1.250
4:40 p. m.....	1.5	6	0.5	0.833
4:53 p. m.....	2.25	13	0.75	0.577

rate upward would be equal to the normal diffusion rate less the effect of gravity and would be slower than normal.

The same glass tube was used as in experiment 30, but instead of being placed horizontally it was placed at an angle of 45 degrees, with the rubber diaphragm on top. The dye was introduced through the diaphragm and diffused much more rapidly downward. The initial diffusion rate was 30 mm. per minute as compared to 10 mm. per minute when the tube was horizontal. A second experiment to determine the diffusion rate upward was performed by injecting from the bottom, and it was found that the initial diffusion rate was only 1.6 mm. per minute as compared with 10 mm. per minute when the tube was horizontal. It is evident from these experiments in the tube that gravity is an important factor in the spread of substances.

To determine if the same thing were true in animals, series 3 was done. A trephine opening was made over each cerebral cortex in the

parietal region, and the occipital bone down to the foramen magnum as well as the first and second cervical vertebrae were removed in order to expose the vermis and cisterna magna. The dye was then injected through a ventricle puncture in the same manner as had been done in the cord. In six animals, series 4 (*a*), the head was supported in a position so that the level of the entire ventricle was higher than the first cervical. In five animals, series 4 (*b*), the head was lowered so that the ventricles were lower than the first cervical. The animal was put in this position prior to the injection and was kept in this position throughout the experiment.

Table 6 shows that when the point of injection (the ventricles) was higher than the first cervical, the average time of appearance of

TABLE 6.—*Effect of Gravity*

Series 4 (*a*)	Experiment Number	Time of Injection Into Left Ventricle	Time of Appearance at Upper Margin of First Cervical	Time of Diffusion in Minutes
	Head Up; Ventricle Higher Than First Cervical			
1	18	2:44:00	2:46:30	2:30
2	19	3:28:30	3:29:30	1:00
3	20	4:10:00	4:16:10	5:10
4	21	3:56:00	3:58:30	2:30
5	22	3:30:00	3:34:00	4:00
6	23	3:13:45	3:18:40	4:55
Average time.	..	.....	.....	3:20
	Head Down; Ventricle Lower Than First Cervical			
Series 4 (*b*)				
7	24	3:21:00	3:29:00	8:00
8	25	4:18:00	4:27:00	9:00
9	26	2:55:00	3:04:00	9:00
10	27	3:53:00	4:01:00	8:00
11	28	3:50:30	3:58:00	7:30
Average time.	..	.....	.....	8:18

the dye was three minutes and twenty seconds. When the point of injection (the ventricles) was lower than the first cervical, the average appearance time was eight minutes and eighteen seconds. All other factors in the two series were identical; one is therefore forced to the conclusion that gravity is one of the most important factors in the spread of substances injected into the cerebrospinal fluid. These conclusions are confirmed by an experiment of Dixon and Halliburton who found that following cisternal injection, when the dog was on his side, the ventricle that lay uppermost was not stained, while the one on the lower side was stained. Their work also bears out our view that there is no directional current between the ventricles and the cisterna, since they found dye in the ventricles following cisternal injection, and we have found it in the cisterna following ventricular injection.

Clinically, it is well known that when a lumbar puncture is done with the patient in the upright position, fluid flows much more freely

than when the patient lies in the horizontal position, and this is in part at least due to gravity.

This involves a fourth factor, namely, the effect of lowering pressure in the subarachnoid space and thus producing an artificial circulation. If the pressure is reduced at one point by nicking the dura, it would be logical to assume that an artificial circulation would be caused by the passage of fluid from areas of higher or normal pressure to that of lowered pressure in an attempt to establish an equilibrium. Howe (1928) stated that reduced pressure would tend to increase the rate of secretion of the cerebrospinal fluid, which would tend to cause a circulation toward the point where the fluid was escaping.

Two dogs were prepared as in series 4. The needle was put into the lateral ventricle, but just before the dye was injected a nick was made in the dura at the atlanto-occipital ligament. In both dogs the dye appeared in less than fifteen seconds at the puncture opening, while with the closed system it took on an average of three minutes and twenty seconds.

This clearly indicates that withdrawing cerebrospinal fluid creates an artificial circulation, and injecting fluids into the lumbar subarachnoid space is an ineffective procedure.

We found that antimeningococcus serum (Parke-Davis) at 21 C. had a specific gravity of 1.024, while our dye mixture had a specific gravity of 1.025. It therefore seems evident that antimeningococcus serum spreads at the same rate of diffusion that the dye did in our experiments.

SUMMARY

1. The evidence brought out in this work shows that the idea that there is a true circulation in cerebrospinal fluid is incorrect.

2. Substances in the cerebrospinal fluid spread by diffusion, but this diffusion is influenced to a great extent by gravity.

3. There is no evidence to show that oscillations in the fluid due to pulse and respiration play any rôle in the movement of cerebrospinal fluid.

4. The reduction of pressure by lumbar puncture creates an artificial circulation toward the point of puncture. This is of great clinical importance. If one desires to inject serum with the idea of having it reach all parts of the central nervous system, it is much more effective to make use of gravity by injecting it into the ventricles or basal cisterna than by injecting it into the lumbar meninges. If one is dealing with meningitis, repeated spinal punctures or permanent lumbar drainage tend to spread the infection by producing an artificial circulation. If drainage is to be employed, it is more effective to do it in the region of the basal cisterna, as recommended by Dandy, than to do it in the lumbar region.

5. In view of the slow rate of diffusion of a substance of the same specific gravity as antimeningococcus serum, it seems advisable, in order to get the greatest effect from the injection of serum, to administer it by cisternal puncture or ventricular puncture unless the process has already spread into the lumbar meninges.

6. Since lumbar puncture is the method usually used for diagnosis, when meningitis is suspected, it should become a routine measure never to withdraw more than a minimal amount.

TABLE 7.—*Diffusion of Dye in Experiment 1*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:58 p. m., injection	...	..	...	.....
4:05 p. m.	1.0	8	1.0	1.250
4:13 p. m.	1.5	8	0.5	0.625
4:23 p. m.	2.0	10	0.5	0.500
4:29 p. m.	2.5	6	0.5	0.833
4:35 p. m.	3.0	6	0.5	0.833
4:56 p. m.	3.0	21	0.0	0.000
Total time	...	59		

TABLE 8.—*Diffusion of Dye in Experiment 2*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:25 p. m., Injection	...	..	...	.....
3:44 p. m.	0.5	19	0.5	0.263
3:51 p. m.	1.3	7	0.8	1.143
3:56 p. m.	1.8	5	0.5	1.000
4:04 p. m.	1.9	8	0.1	0.125
4:10 p. m.	2.0	6	0.1	0.166
4:43 p. m.	2.0	33	0.0	0.000
Total time	...	78		

PROTOCOLS

SERIES 1.—*Experiment 1.*—Medium-sized dog. Laminectomy, first dorsal to fifth lumbar vertebra. Injected 0.5 cc. of dye at level of the third lumbar vertebra.

Experiment 2.—Small dog. Laminectomy, first sacral to third cervical vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

Experiment 3.—Large dog. Laminectomy, first cervical to tenth dorsal vertebra. Very little hemorrhage. Injected 0.5 cc. of dye at level of the first dorsal vertebra, needle pointing up the cord. At the end of injection the dye margins were at the fifth cervical and fourth dorsal vertebra.

Experiment 4.—Small dog. Took anesthetic well. Bones very vascular and soft. Marked pulsations of dye margin with respiration extent from 1 to 2 cm. Laminectomy, fifth lumbar to first thoracic vertebrà. Injected 0.5 cc. of dye at the level of the third lumbar.

Experiment 5.—Medium-sized dog. Laminectomy from fourth lumbar to second dorsal vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra. An air bubble preceded the dye margin and apparently acted mechanically in preventing diffusion of the dye.

Experiment 6.—Young, medium-sized collie. Took anesthetic with difficulty. Bones soft and vascular. Dog of hypersthenic type. Laminectomy, first sacral to first dorsal vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

Experiment 7.—Medium-sized, young dog. Took anesthetic well. Bones soft. Hypersthenic type. Laminectomy, fifth lumbar to second dorsal vertebra; 0.5 cc. of dye injected at the level of the third lumbar vertebra.

TABLE 9.—*Diffusion of Dye in Experiment 3*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:47 p. m., injection	...	..	...	.....
3:50 p. m.	2.7	3	2.7	9.000
3:55 p. m.	4.0	5	1.3	2.600
4:00 p. m.	4.1	5	0.1	0.200
4:05 p. m.	4.1	5	0.0	0.000
Total time	...	18		

TABLE 10.—*Diffusion of Dye in Experiment 4*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:01 p. m., injection	...	..	...	.....
3:08 p. m.	1.4	7	1.4	2.000
3:12 p. m.	1.8	4	0.4	1.000
3:14 p. m.	2.4	2	0.6	3.000
3:17 p. m.	2.8	3	0.4	1.333
3:22 p. m.	3.1	5	0.3	0.600
3:26 p. m.	3.3	4	0.2	0.500
3:30 p. m.	3.4	4	0.1	0.250
3:34 p. m.	3.4	4	0.0	0.000
Total time	...	33		

TABLE 11.—*Diffusion of Dye in Experiment 5*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute	Remarks
3:25 p. m., injection	...	..	...	.....	Air bubble injected before dye
4:02 p. m.	1.5	10	1.5	1.500	
4:07 p. m.	1.5	5	0.0	0.000	
4:17 p. m.	1.5	10	0.0	0.000	
4:30 p. m.	1.5	13	0.0	0.000	
Total time	...	38			

Experiment 8.—Old, small dog. Asthenic in type. Bones very hard. Even skin was overly vascular and hemorrhage was profuse even from skin incision. Hemorrhage hard to control throughout operation. Laminectomy, first sacral to first dorsal vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

Experiment 9.—Young, medium-sized dog. Hypersthenic in type. Bones soft. Hemorrhage well controlled. Laminectomy, first sacral to second dorsal vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

Experiment 10.—Fairly young, medium-sized dog, hypersthenic in type. Bones fairly soft. Little hemorrhage. Laminectomy, fifth lumbar to first dorsal vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

TABLE 12.—*Diffusion of Dye in Experiment 6*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:39 p. m., injection	...	..	...	.....
3:43 p. m.	1.0	4	1.0	2.500
3:51 p. m.	2.0	8	1.0	1.250
3:56 p. m.	2.5	5	0.5	1.000
4:08 p. m.	2.8	12	0.3	0.250
4:13 p. m.	2.9	5	0.1	0.200
4:17 p. m.	2.9	4	0.0	0.000
Total time	...	38		

TABLE 13.—*Diffusion of Dye in Experiment 7*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:45 p. m., injection	...	..	...	.....
3:57 p. m.	1.0	12	1.0	0.833
4:15 p. m.	2.0	18	1.0	0.555
4:22 p. m.	2.5	7	0.5	0.714
4:30 p. m.	3.0	8	0.5	0.625
4:45 p. m.	3.0	15	0.0	0.000
Total time	...	60		

TABLE 14.—*Diffusion of Dye in Experiment 8*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:10 p. m., injection	...	..	...	.....
3:19 p. m.	1.0	9	1.0	1.111
3:36 p. m.	2.0	17	1.0	0.588
3:50 p. m.	3.0	14	1.0	0.714
3:55 p. m.	3.1	5	0.1	0.200
4:00 p. m.	3.1	5	0.0	0.000
Total time	...	50		

TABLE 15.—*Diffusion of Dye in Experiment 9*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:44 p. m., injection	...	..	...	.....
4:00 p. m.	3.0	16	3.0	1.875
4:15 p. m.	5.8	15	2.8	1.866
4:36 p. m.	10.0	21	4.2	2.000
4:40 p. m.	10.1	4	0.1	0.250
4:45 p. m.	10.1	5	0.0	0.000
Total time	...	61		

Experiment 11.—Young pup, hypersthenic in type. Bones soft and vascular. Considerable hemorrhage even from the skin. Took anesthetic well. Laminectomy, fifth lumbar to first dorsal vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

Experiment 12.—Large dog. Sthenic in type. Took anesthetic well. Considerable hemorrhage. Laminectomy, first sacral to twelfth dorsal. Bilateral trephine over parietal regions. Injected 0.5 cc. of dye at the level of the third lumbar vertebra. Injection time 10:32 a. m.

The dura under the trephine openings was examined every half hour for appearance of the dye beneath it. The head of the dog was maintained at a level so that the center of the ventricles was level with the spinal cord.

TABLE 16.—*Diffusion of Dye in Experiment 10*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:13 p. m., injection.....	...	..	...	.....
3:25 p. m.....	3.0	12	3.0	2.500
3:40 p. m.....	6.0	15	3.0	2.000
3:55 p. m.....	9.5	15	3.5	2.333
4:00 p. m.....	9.5	5	0.0	0.000
Total time..	...	47		

TABLE 17.—*Diffusion of Dye in Experiment 11*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:43 p. m., injection.....	...	..	...	.....
4:00 p. m.....	1.5	17	1.5	0.882
4:10 p. m.....	2.0	10	0.5	0.500
4:27 p. m.....	2.1	17	0.1	0.058
4:30 p. m.....	2.1	3	0.0	0.000
Total time.....	...	47		

TABLE 18.—*Diffusion of Dye in Experiment 13*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute	Pulse (Femoral) Rate	Respiration Rate	Remarks
4:51 p. m., injec.	...	..	...	.....	135	36	
4:53 p. m.....	2.3	2	2.0	10.000	141	42	
4:56 p. m.....	3.0	3	1.0	3.333	138	49	
5:00 p. m.....	9.2	4	6.2	15.500	159	48	Spastic dog; cord at a slope of 10 degrees downward

At the end of ten hours no dye had appeared at the level of the trephine openings. The dog was killed and an autopsy was performed.

SERIES 2.—*Experiment 13.*—A large asthenic dog. Took anesthetic only fairly well. Much hemorrhage. Laminectomy, fifth lumbar to first dorsal vertebra. Inserted cannula into left femoral for kymographic record of pulse rate. Used bag for respiration rate record. Dog was spastic when injection was made and only very lightly under the anesthetic. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

Experiment 14.—Large dog. Laminectomy, fifth lumbar to first dorsal vertebra. Hypersthenic dog, very broad back. Hemorrhage well controlled. Took anesthetic fairly well. Inserted cannula into left femoral. Used bag for res-

piration rate. Injected 0.5 cc. of dye at the level of the third lumbar vertebra. Dog well under anesthetic at the time of injection.

Experiment 15.—Medium-sized dog. Took anesthetic well. Little hemorrhage until cannula came out of femoral after second reading had been made. Cannula was immediately reinserted. Laminectomy, first sacral to first dorsal vertebra. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

TABLE 19.—*Diffusion of Dye in Experiment 14*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute	Pulse (Femoral) Rate	Respiration Rate
3:51 p. m., injection...		..	...	.....	133	17
3:55 p. m.....	3.5	4	3.5	8.750	141	57
3:59 p. m.....	4.5	4	1.0	2.500	174	63
4:03 p. m.....	5.0	4	0.5	1.250	147	63
4:09 p. m.....	6.0	6	1.0	1.666	165	48
4:16 p. m.....	6.2	7	0.2	0.285	168	48
4:25 p. m.....	6.4	9	0.2	0.222	156	51

TABLE 20.—*Diffusion of Dye in Experiment 15*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute	Pulse (Femoral) Rate	Respiration Rate
3:45 p. m., injection...		..	...	.....	150	21
3:54 p. m.....	2.5	9	2.5	2.777	150	24
4:00 p. m.....	4.0	6	1.5	1.666	180	33
4:05 p. m.....	4.4	5	0.4	0.800	...	42

TABLE 21.—*Diffusion of Dye in Experiment 16*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute	Pulse (Femoral) Rate	Respiration Rate
3:33 p. m., injection...		..	...	.....	135	60
3:36 p. m.....	1.6	3	1.6	5.333	168	66
3:48 p. m.....	6.0	12	4.4	3.666	174	48
3:53 p. m.....	8.5	5	2.5	5.000	...	..
3:54 p. m.....	8.5	1	0.0	0.000	150	60
3:59 p. m.....	9.0	5	0.5	1.000	168	45
4:08 p. m.....	10.5	9	1.5	1.666	36	36
4:24 p. m.....	11.0	16	0.5	0.312	...	..

Experiment 16.—Large dog. Fairly well controlled hemorrhage. Took anesthetic well. Laminectomy, first sacral to first dorsal vertebra. Inserted cannula into left femoral. Used bag for respiration rate. Injected 0.5 cc. of dye at the level of the third lumbar vertebra.

SERIES 3.—*Experiment 17.*—Large dog. Laminectomy, first cervical to tenth dorsal vertebra. Very little hemorrhage. Injected 0.5 cc. of dye at the level of the first dorsal vertebra, needle pointing up the cord. At the end of injection, the dye margins were at the fifth cervical and fourth dorsal vertebra.

SERIES 4 (*a*).—*Experiment 18.*—Bilateral trephine for ventricular puncture. Laminectomy, first cervical to eighth dorsal vertebra. Injected 0.5 cc. of dye into the left ventricle. Ventricles higher than first cervical. Injected into

ventricle at 2:44 p. m. Dye appeared at upper margin of first cervical at 2:46 p. m. Spread down to fifth cervical at 4:37 p. m.

Experiment 19.—Bilateral trephine for ventricular puncture. Laminectomy, first cervical to sixth dorsal vertebra. Injected 0.5 cc. of dye into the left ventricle. Ventricles higher than first cervical. Injected into ventricle at 3:28 p. m. Dye appeared at upper margin of first cervical at 3:29 p. m., spread down the cord. Right ventricle was punctured at 4:04 p. m.; first drop showed color; at 4:05 p. m. the dura at the fifth dorsal was punctured and color came down the cord to the point of reduced pressure and flowed out of the puncture hole.

Experiment 20.—Small dog. Bilateral trephine, for ventricular puncture. Laminectomy, first cervical to first dorsal. Injected 0.5 cc. of dye subdurally over the cortex at 3:42 p. m.; at 3:52 p. m. no dye had appeared at the first cervical. Left ventricle was punctured and fluid was clear. Hole sealed. Withdrew 2.5 cc. of clear fluid at the level of the first cervical subdurally at 4:05 p. m. At 4:05 p. m., a bilateral trephine 2.2 cm. in front of the original openings was performed. At 4:10 p. m., 0.5 cc. of dye was injected into the

TABLE 22.—*Diffusion of Dye in Experiment 17*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute	Remarks
Diffusion Up the Cord					
3:45 p. m., injection ...		..	...	.....	At end of injection at first
3:50 p. m.....	2.7	3	2.7	9.000	dorsal, dye margin was at
3:55 p. m.....	4.0	5	1.3	2.600	fifth cervical
4:00 p. m.....	4.1	5	0.1	0.200	
Diffusion Down the Cord					
3:45 p. m., injection ...		..	...	.....	At end of injection at first
3:50 p. m.....	2.8	3	2.8	9.333	dorsal, dye margin was
3:55 p. m.....	4.1	5	1.3	2.600	at fourth dorsal
4:00 p. m.....	4.2	5	0.1	0.200	

left ventricle. Dye appeared at 4:16 p. m. at the upper margin of the first cervical. Throughout the experiment the ventricles were higher than the level of the first cervical.

Experiment 21.—Small dog. Hemorrhage easily controlled. Took anesthetic well. Bilateral trephine for ventricular puncture. Injected 0.5 cc. of dye into left ventricle at 3:56 p. m. after several attempts to hit the ventricle had been made. Dye appeared at the upper margin of the first cervical at 3:58:30 p. m. Ventricles higher than the level of the first cervical.

Experiment 22.—Medium-sized dog. Hypersthenic in type. Very little hemorrhage. Very good exposure. Bilateral trephine and first and second cervical laminectomy and occipital craniotomy for exposure of the vermis and lateral lobes of the cerebellum. Injected 0.5 cc. of dye into the left ventricle at 3:30 p. m.; at 3:31 p. m. it could be seen in the cisterna and appeared at the upper margin of the first cervical at 3:34 p. m. There was a definite line of demarcation at the base of the vermis and the dye did not go up over the vermis or cerebellum. No dye appeared over the right cortical area of the cerebrum and none was obtained on nicking the dura at 3:35 p. m., the first drop being clear. Ventricles higher than first cervical.

Experiment 23.—Small dog. Difficult anesthetic. Bilateral trephine for ventricular puncture. Laminectomy of first and second cervical vertebra. Occipital

craniotomy for exposure of the vermis and lateral lobes of the cerebellum. Injected 0.5 cc. of dye into left ventricle at 3:13:45 p. m. Appeared at upper margin of first cervical at 3:18:40 p. m. Dye did not go up over the vermis. None appeared on the first drop when the dura of the right cortical area of the cerebrum was nicked, at 3:20 p. m. Ventricles higher than the first cervical.

SERIES 4(*b*).—*Experiment 24.*—Old dog. Bilateral trephine for ventricular puncture; first cervical laminectomy. Very little hemorrhage. Injected 0.5 cc. of dye into the left ventricle at 3:21 p. m. Dye appeared at upper margin of the first cervical at 3:29 p. m. The level of the ventricles was lower than the level of the first cervical. No dye appeared over the right cerebral cortex area at 3:45 p. m.

Experiment 25.—A young, large dog. Hemorrhage well controlled. Took anesthetic well. Bilateral trephine for ventricular puncture. First and second cervical laminectomy. Injected 0.5 cc. of dye into left ventricle at 4:18, p. m., appeared at upper margin of the first cervical at 4:27 p. m. Level of the ventricles lower than the level of the first cervical. No dye appeared over the right cerebral cortex area at 5:00 p. m.

Experiment 26.—Large dog. Took anesthetic poorly. Much hemorrhage. Bilateral trephine and first cervical laminectomy and occipital craniotomy to expose vermis and lateral lobes of the cerebellum. Posterior portion of foramen magnum removed. Injected 0.5 cc. of dye into the left ventricle at 2:55 p. m. Appeared at upper margin of the first cervical at 3:04 p. m. Level of the ventricles was lower than the level of the first cervical. No dye appeared at the opening over the right cerebral cortex at 3:15 p. m.

Experiment 27.—Medium-sized dog. Took anesthetic well. Bilateral trephine and first and second cervical laminectomy. Removed posterior part of foramen magnum and exposed vermis. Very little hemorrhage. Excellent exposure. Injected 0.5 cc. of dye into the left ventricle at 3:53 p. m. Dye appeared at upper margin of the first cervical at 4:01 p. m. Level of the ventricles was lower than the first cervical. No dye appeared over the vermis and none subdurally over the right cerebral cortex at 4:30 p. m.

Experiment 28.—Very large dog. Difficult anesthetic. Average amount of hemorrhage. Bilateral trephine and first and second cervical laminectomy. Removed posterior part of foramen magnum and occipital bone to expose vermis and part of the lateral lobes of the cerebellum. Injected 0.5 cc. of dye into the left ventricle at 3:50:30 p. m. Dye appeared at the upper margin of the first cervical at 3:58:00 p. m. Level of the uppermost part of the ventricles was lower than the level of the first cervical as done in the entire series 4(*b*). The dog was killed at 4:00 p. m. and the brain case removed. The dye had diffused along the lower portion of the cerebrum but did not reach the top of the cortex, under the trephine opening on the right side or on the left side.

Experiment 29.—Large dog. Took anesthetic fairly well. Very little hemorrhage. Bilateral trephine and laminectomy, first sacral to twelfth dorsal vertebra. 0.5 cc. of dye injected into the left ventricle at 11:27 a. m.

The dura in the lumbar region was inspected every half hour for appearance of the dye beneath it. The head of the dog was maintained at a level so that the center of the ventricles was level with the spinal cord.

At the end of ten hours no dye had appeared at the lumbar exposure. The dog was killed and autopsied.

SERIES 5.—*Experiment 30.*—A glass tube 5 mm. in diameter and 30 cm. long was sealed at one end and completely filled with cerebrospinal fluid. The other

end was covered with a rubber diaphragm. With the tube in the horizontal position, tested by a spirit level, dye was injected through the diaphragm by the same technic used on the dogs.

Experiment 31.—Effect of gravity in vitro. Tube at angle of 45 degrees; 0.5 cc. of trypan-blue injected at the top.

Experiment 32.—Tube at an angle of 45 degrees; 0.5 cc. of trypan-blue injected at the bottom.

SERIES 6.—*Experiment 33.*—Large dog. Took anesthetic very well. Very little hemorrhage. Bilateral trephine and laminectomy, first sacral to twelfth

TABLE 23.—*Diffusion of Dye in Experiment 30*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
5:13 p. m., injection	...	..	...	.....
5:14 p. m.	1.0	1	1.0	10.000
5:18 p. m.	2.0	4	1.0	2.500
5:28 p. m.	3.0	10	1.0	1.000
5:48 p. m.	4.0	20	1.0	0.500

TABLE 24.—*Diffusion of Dye, Down, in Experiment 31*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
3:53 p. m., injection	...	..	...	.....
3:55 p. m.	6	2	6	30:000
3:56 p. m.	10	1	4	40:000
3:57 p. m.	13	1	3	30:000
3:58 p. m.	15	1	2	20:000

TABLE 25.—*Diffusion of Dye, Up, in Experiment 32*

Time	Diffusion in Centimeters from Initial Point	Time Interval in Minutes	Centimeters Diffused per Time Interval	Diffusion Rate in Millimeters per Minute
4:25 p. m., injection	...	..	...	.....
4:28 p. m.	0.5	3	0.5	1.666
4:34 p. m.	1.0	4	0.5	1.250
4:40 p. m.	1.5	6	0.5	0.833
4:53 p. m.	2.25	13	0.75	0.577

dorsal vertebra. Injected 0.5 cc. of dye into the subdural space over the parietal region of the cerebral cortex at 12:02 p. m. The dura in the lumbar region was inspected every half hour for appearance of the dye beneath it. The head of the dog was maintained at a level so that the center of the ventricles was level with the spinal cord. At the end of ten hours no dye had appeared at the lumbar exposure. The dog was killed and autopsied.

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MANIC-DEPRESSIVE PSYCHOSIS AS SEEN IN PRIVATE PRACTICE

SEX DISTRIBUTION AND AGE INCIDENCE OF FIRST ATTACKS *

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Published descriptions of manic-depressive psychosis are based mainly on the observation of patients in hospitals or sanatoriums for the insane. This type of material is incomplete; it represents only that fraction of patients with manic-depressive psychosis whose condition is so serious as to necessitate commitment. The vast numbers of patients with this psychosis who are afflicted so mildly that hospitalization is not necessary have influenced little, if at all, descriptions of this disorder. The material used for this study consists of 633 cases of manic-depressive psychosis from the private records of Dr. Hugh T. Patrick. These cases have an obvious advantage for study; not only do they represent cases in which the patients were so ill that institutionalization was imperative, but in this series there are a great number of patients who were so mildly afflicted that hospitalization was not necessary. A study of such material gives a view of the disorder which is different from that obtained from patients in hospitals. These private cases were studied from the standpoint of sex distribution and age of onset of the first attack. The data so obtained were compared with similar data in the literature, and interesting and striking differences were found. These differences constitute the subject of this report.

SEX

Statistics as to the sex distribution of manic-depressive psychosis vary slightly, but all authors agree that it is predominantly a disease of the female. Kraepelin [1] stated that 70 per cent are females; Bleuler [2] gave 70 per cent; Pilcz,[3] 66.6 per cent, and Strohmayer,[4] 75 per cent.

* Submitted for publication, April 15, 1929.
* Read at a meeting of the Chicago Neurological Society, March 21, 1929.
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1. Kraepelin, E.: Manic-Depressive Insanity and Paranoia, translated by Mary Barclay, Edinburgh, E. & S. Livingstone, 1921.
2. Bleuler, E.: Textbook of Psychiatry, translated by A. A. Brill, New York, The Macmillan Company, 1924.
3. Pilcz, A.: Psychoses maniaques dépressives et periodiques, in Traité international de psychologie pathologique, Paris, Félix Alcan, 1911, vol. 2.
4. Strohmayer: Manisch-Depressive Irresein, Wiesbaden, F. C. W. Vogel, 1914.

Stransky [5] stated that in women there is a decided predilection for this disorder. Soukhanoff and Gannouchkine [6] found that 75 per cent of their patients were female. Dercum [7] stated that females predominate in the ratio of 2:1. Henderson and Gillespie [8] stated that it is estimated that 70 per cent of the patients are female. Gordon [9] found that of 210 patients 150 were female, a percentage of 71.4. Durand [10] stated that 63 per cent are female. In the state hospitals of New York [11] for seventeen years, ending in 1925, there were received as first admissions 8,611 female and 5,045 male patients with this psychosis, or 63.1 per cent female and 36.9 per cent male. In Illinois [12] for four years, ending in 1927, there were received as first admissions 592 females and 411 males with this disorder, or 59.1 per cent female and 40.9 per cent male.

Analysis of the present series shows no such preponderance of female patients. Among 633 patients 325 were female and 308 male —51.4 per cent female and 48.6 per cent male. Even this slight difference is minimized by the fact that in the mixed metropolitan population there is a predominance of females in the ratio of 50.3 to 49.7 per cent. It appears, then, from a study of this series that manic-depressive psychosis is not preponderantly a disease of the female.

AGE

Age Incidence of the First Attack.—Eschle [13] stated that two thirds of the patients have the onset before the age of 25; Bleuler,[2] that in the majority of cases the disease starts between the ages of 15 and 30, and

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Ziehen,[14] that in most cases the disorder begins between 15 and 35. Dercum [7] stated that this psychosis begins most frequently between 18 and 30; Pilcz [3] and Stransky [5] stated that there is a special predilection for first attacks around puberty and the climacteric, and Drysdale [15] stated that in most cases the first attack occurs before 25. Buckley [16] wrote that there appears to be no age of selection, and that in the majority of cases the first attack appears between 25 and 40; and Durand [9] that the maximum frequency of first attacks is between 15 and 28 in females and between 18 and 30 in males. Kraepelin,[1] in a study of 903 hospital cases, found that the greatest frequency of first attacks was between 15 and 20. Regarding the age incidence, Kraepelin's statistics are the most comprehensive that I can find, and may be used

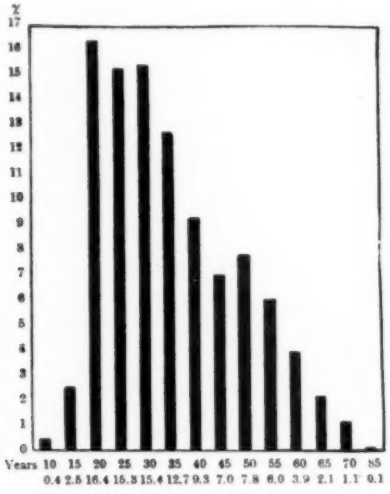

Chart 1.—Distribution of first attacks of manic-depressive psychosis, according to age, in the 903 cases of Kraepelin's series. From Kraepelin's Manic-Depressive Insanity and Paranoia, and reproduced by permission of the publishers, E. & S. Livingstone, Edinburgh.

as a basis of comparison. In Kraepelin's series (chart 1), more first attacks occurred between the ages of 15 and 20 than in any other half decade. In the next five years there is a slight decline, and between 25 and 30 another slight rise; then there is a continuous decline in the incidence of first attacks with the exception of a slight rise between 45 and 50.

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Age Distribution of the First Attack in My Series.—This could be determined in 615 cases. The results appear in chart 2. There was a steady increase in the number of first attacks until the age of 30. More first attacks appeared between 26 and 30 (18.4 per cent) than in any other five year period. This is a full decade later than the maximum incidence of first attacks in Kraepelin's series. After 30

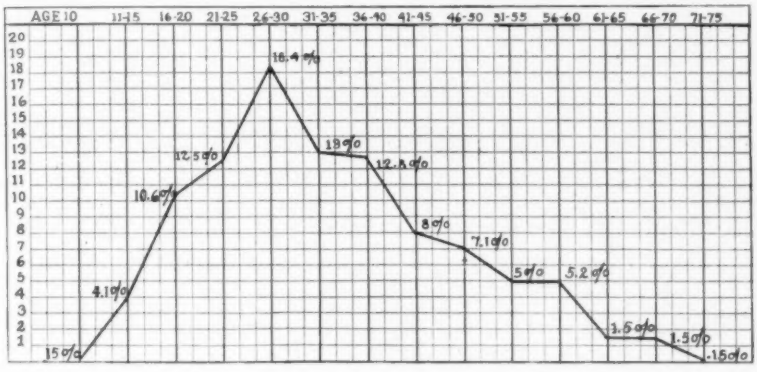

Chart 2.—Distribution of first attacks according to age in 615 patients of both sexes.

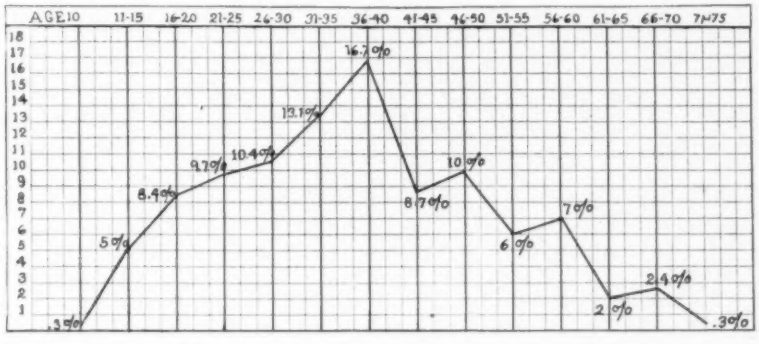

Chart 3.—Distribution of first attacks, according to age, in 299 male patients.

there was a gradual decline, but more first attacks appeared between 31 and 35 (13 per cent) and between 36 and 40 (12.8 per cent) than appeared between 16 and 20 (10.6 per cent) or between 21 and 25 (12.5 per cent). In this series only 27.3 per cent of first attacks appeared before 25, and 45.7 per cent occurred before 30; in other words, in this series the majority of first attacks appeared after the age of 30.

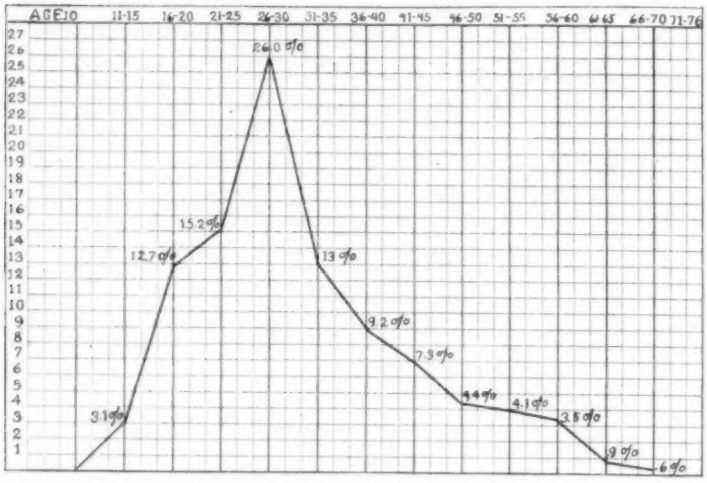

Chart 4.—Distribution of first attacks, according to age, in 316 female patients.

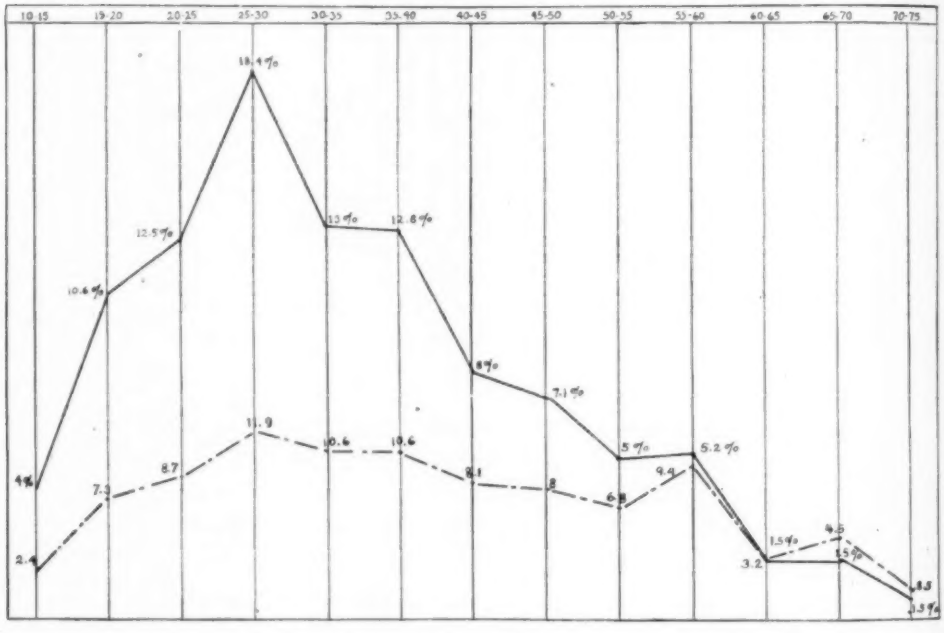

Chart 5.—Frequency of first attacks in relation to percentage of general population at different ages. The solid line indicates the percentage of distribution by age, and the broken line the number of first attacks per 1 per cent of the general population, also according to age. The figures along the top of the chart indicate age in years.

Age Distribution of First Attack According to Sex.—In the male series, comprising 299 cases (chart 3), there was a steady increase in the number of first attacks until the age of 40. Between 36 and 40 there were more first attacks than in any other five year period (16.7 per cent). After 40 there was a gradual decline, with slight increases between 46 and 50 and between 56 and 60. In the male series, 66.2 per cent of first attacks occurred after 30.

TABLE 1.—*Age Distribution of First Attack of Manic-Depressive Psychosis According to Sex*

	Male		Female		Both Sexes	
Age, Years	Number	Per Cent	Number	Per Cent	Number	Per Cent
To 10	1	0.3	0	0.0	1	0.15
11-15	15	5.0	10	3.1	25	4.10
16-20	25	8.4	40	12.7	65	10.60
21-25	29	9.7	48	15.2	77	12.50
26-30	31	10.4	82	26.0	113	18.40
31-35	39	13.1	41	13.0	80	13.00
36-40	50	16.7	29	9.2	79	12.80
41-45	26	8.7	23	7.3	49	8.00
46-50	30	10.0	14	4.4	44	7.10
51-55	18	6.0	13	4.1	31	5.00
56-60	21	7.0	11	3.5	32	5.20
61-65	6	2.0	3	0.9	9	1.50
66-70	7	2.4	2	0.6	9	1.50
71-75	1	0.3	0	0.0	1	0.15

TABLE 2.—*Frequency of First Attacks per 1 Per Cent of Population by Half Decades*

Age, Years	Percentage of Distribution Among General Population, 1920	Number of First Attacks	Percentage	Frequency Value Number of First Attacks, per 1 per Cent of Population
10-15	10.1	25	4.00	2.40
15-20	8.9	65	10.60	7.30
20-25	8.8	77	12.50	8.70
25-30	8.6	113	18.40	11.90
30-35	7.6	80	13.00	10.60
35-40	7.4	79	12.80	10.60
40-45	6.0	49	8.00	8.10
45-50	5.5	44	7.10	8.00
50-55	4.5	31	5.00	6.80
55-60	3.4	32	5.20	9.40
60-65	2.8	9	1.50	3.20
65-70	2.0	9	1.50	4.50
70-75	1.3	1	0.15	0.85

In the female series, 316 cases (chart 4), there was a sharp rise until 30. More first attacks occurred between 26 and 30 (26 per cent) than in any other five year period, and more first attacks occurred between 31 and 35 (13 per cent) than between 16 and 20 (12.7 per cent). After 30 there was a steady decline. In the female series, 69 per cent of first attacks occurred after 25 and 43 per cent after 30.

The data of all three series are given in table 1.

The next question is whether the age incidence of first attacks is in relation to the age of the patient or to the percentage at different ages

of the general population. To determine this, the percentage of population by half decades was obtained from the census of 1920. Then I determined the number of first attacks per 1 per cent of population in each half decade. The result is the frequency value for each half decade (table 2). This shows that first attacks of manic-depressive psychosis are relatively more rare between 15 and 20 than in any subsequent period except after 50, and that as the curve of age incidence of first attacks rises and falls, so does that of age incidence in the general population, though not so sharply.

CONCLUSIONS

Six hundred and thirty-three extramural cases of manic-depressive psychosis were studied for sex distribution, and six hundred and fifteen for age distribution of first attacks. The ratio of female to male patients in this series was 51.4 to 48.6 per cent. In the entire series, first attacks were most common between 26 and 30; in the male series, between 36 and 40, and in the female series, between 26 and 30. In the entire series, 54.3 per cent of first attacks occured after 30; in the male series, 66.2 per cent after 30, and in the female patients, 43 per cent after 30. The difference between these observations and those of others is in all likelihood due to the fact that other studies or statements are based on patients in hospitals, and only the patients with the most severe cases are hospitalized; the present report is based on a study of extramural patients. It would seem, then, that the predominance of younger patients and of female patients in other studies is due to the fact that manic-depressive psychosis runs a more malignant course in younger persons and in females, since in hospital statistics these are much more prevalent than in this series.

News and Comment

SECOND INTERNATIONAL CONGRESS FOR SEX RESEARCH

The Second International Congress for Sex Research will be held in the House of the British Medical Association, Tavistock Square, London, August 3 to 9, 1930, under the presidency of Professor F. A. E. Crew, of Edinburgh. Both in membership and in importance it is expected that this meeting will notably excel the First International Congress, held in Berlin in October, 1926. Those who are interested are requested to write Professor Crew, The University, West Mains Road, Edinburgh.

The organization and purposes of the congress can be fully served only by having all of the many aspects of sex research properly represented among the papers read at the London meeting. To American workers it is highly desirable that American investigations and investigators should be adequately represented at London, particularly since few persons from this country attended the first congress.

An American committee has been formed for the purpose of securing a maximum participation in the congress by workers in this country. The members of this committee, representing biology, medicine, psychology, sociology and anthropology, are: Dr. Oscar Riddle, chairman, Dr. A. A. Brill, Dr. Calvin P. Stone, Dr. William F. Ogburn and Dr. Clark Wissler.

AMERICAN ORTHOPSYCHIATRIC ASSOCIATION

The Seventh Annual Meeting of the American Orthopsychiatric Association will be held at the Hotel Pennsylvania, New York, on Feb. 21 and 22, 1930. The president is Dr. Lawson G. Lowrey, and the secretary, Dr. George S. Stevenson, 370 Seventh Ave., New York.

Abstracts from Current Literature

AFFECTIVITY AND PSYCHOTHERAPY. O. L. FOREL, J. f. Psychol. u. Neurol. **38**:58, 1929.

This paper is the author's contribution to the Forel-Festschrift in honor of August Forel's eightieth birthday. O. L. Forel's formulations may be briefly summarized as follows: Affectivity is that form of sensibility which one designates subjectively as "feeling." It is a general attribute of living substance, and therefore it is useless to attempt to construct a localization for it. Because the nervous system has phylogenetically acquired to a certain degree a monopoly of sensibility, the existence of an individual sensibility peculiar to the various organs cannot be excluded. Not only do the endocrine glands, sympathetic system and especially the various organs of the body participate in affectivity, but what is of paramount importance is the reaction of the "psychic" on the "physiologic." These interactivities, constant as they are, can be appreciated only when one disregards the conception of the artificially created dualism — the opposition between "psychic" and "physical."

The division between psychic, chemical and physical should no longer mislead one into verbal and conceptual speculations, because these also hinder clinical progress. As an example of this is cited the experience of every psychiatrist who is confronted with the similarities and differences between genuine (chemical?) melancholia, presenile (organic?) and so-called psychogenic depression. These three may resemble each other so closely and run so similar a course that it is actually impossible to determine which type of depression one may be dealing with. That in a doubtful case the active therapeutist always treats the patient as if the psychogenic component was a complicating factor or the principal factor, is not surprising when one takes into consideration that in psychiatry as in medicine, in general, treatment "ex juvantibus" is so frequently indicated. Similarly, in a case of disturbance of the sympathetic nervous system which has made its first appearance following a psychogenic trauma, and is therefore regarded as secondary in nature, one is certain to achieve better results earlier when in addition to the medicinal treatment a thorough psychic approach to the problem is also undertaken. And so, in a case of exophthalmic goiter appearing soon after a psychic shock it is also most advantageous to direct some therapeutic effort to the psyche at the same time that the patient is being treated for disturbances in the body chemistry.

One is too readily misled and unjustifiably satisfied with himself when, in employing psychotherapy, he believes that it is purely his own personality that affects favorably the patient's psyche. While it may be true that in many cases the psychotherapist's personality may play a significant rôle in the patient's tendency to transference and thus bring about more or less curative results, much better and more permanent results can be expected when the physician-patient relationship does not become too close but reaches a point at which the affectivity of the patient may spread itself out and radiate in all conceivable directions. Individual analysis, desirable as it may be, must never replace collective thinking, because biologically one is shackled to the surrounding world by thousands of chains, and it is only a diseased mind that loses contact with the rhythm of life. The contact between physician and patient must serve only as a preliminary step — as a bridge, the crossing of which will allow the patient's return to the reality of life.

That sexual errors and conflicts are at the basis of almost every neurosis seems today to be an almost generally accepted fact. Who knows but that this conception may be changed tomorrow and this almost universally accepted causal relationship between sex and neurosis be denied, or that it may be found that interpretations were too hasty and that symptoms were erroneously considered

causative factors? As far as the author is concerned, he is inclined to look on the individual sex instinct only as one link in the collective ties that have a tendency to preserve the species, so that to him sexual-neurotic reactions merely represent partial manifestations of a neurotic psyche in its reactions to the outside world. It is certain that the individual does not ponder over the where and whence of his instincts; he merely looks blindly for the pleasures; in the course of this groping in the dark it is to be expected that he must stumble over some difficulties, some obstructions which are at the basis of social life and the necessary protective measures that insure a proper family, state and cultural existence. In this connection it must, however, also be borne in mind that these obstructions and inhibitions are themselves a product of social instincts and are just as "normal" as the pleasures. The everlasting conflict between individualism and collectivity is in itself healthful and stimulating; it is only when this conflict gives rise to a neurosis that the physician must "insert" himself, even if only temporarily, into the psychic life of the patient representing to him the outside world.

Taking into consideration the numerous phases and shades of life's affect, one cannot help but be astonished at the rôle played by experiences in community life. As a matter of fact, there are a great number of antitheses in life's affect that can be aroused only through collective experiences. Take, for instance, the love for truth and lying, sincerity and hypocrisy, bravery and cowardice, love and hatred, egoism and altruism, etc. One would almost be tempted to assume that a man living in solitude would have to develop a frightfully necessary affectivity because life's affect becomes differentiated only through the individual variations of fellow-beings and through social interdependence.

It is for these and similar reasons that August Forel turned his activities from brain research to psychiatry and later to sociology; to him, clinical limitations were too narrow and too artificial.

The investigation of the healthy as well as of the diseased mind cannot be carried out far enough psychologically; purely clinical investigation is too artificial. Analysis will not accomplish everything. The liberation of the patient from his symptoms demands thorough syntheses by reinserting him into the biologic rhythm and by reestablishing the lost equilibrium between his ego and the world around him. The psychobiologic conception of the world and psychically free man tower above the egoistic conflicts that are hidden in the very germ plasm of two persons, above the family spirit, above chauvinistic nationalism, above all the enclosing barricades of the affect which imprison the thousands of conflicts harboring within one's soul, and above everything that pains one and embitters one.

KESCHNER, New York.

LOCAL VULNERABILITY. W. SPIELMEYER, Ztschr. f. d. ges. Neurol. u. Psychiat. **118**:1 (Dec.) 1928.

Spielmeyer discusses the causative factors behind the particular seat and spread of pathologic processes. The problem is important not only for those diseases which attack a certain definite site in the nervous system, but also in widespread processes which affect one part or another predominantly. Such is the case in paresis in which, though the process is widespread, the frontal lobes are most affected, while the occipital are little involved. There are also certain diseases which, though usually generalized, sometimes attack one part of the nervous system in particular. Such is the case in amaurotic idiocy which is usually a generalized disease of the ganglion cells, but which in the late infantile form of Bielschowsky attacks the cerebellum in particular. The system diseases, the extrapyramidal diseases and those of the gray nuclei are further examples of selection in diseases of the nervous system.

What are the factors which cause the particular localization of disease processes? Why have certain processes a predilection for certain places or a certain electivity? Spielmeyer says, in answer to these questions, that pathogenetic analysis reveals no one single factor as a cause in the localization of disease in the nervous system.

There are many factors, known and unknown, simple and complicated. The factors known today are the systemic, the circulatory and the spinal fluid factors.

Although there is a good deal of objection to the concept of a system disease which involves certain parts of the nervous system by preference, Spielmeyer subscribes to the view that such system diseases exist in this manner. Certain nervous mechanisms are particularly susceptible to disease, and many noxious agents have an affinity for definite parts of the nervous system. The Purkinje cells of the cerebellum are susceptible to disease. The nucleus dentatus is particularly susceptible in typhus fever, and Spielmeyer believes that the noxious agent has produced here a system disease. The same is true of many other infections and intoxications. Ehrlich demonstrated the existence of such a system vulnerability. In the elective affinity of a substance for certain organs and systems he saw a function of its chemical constitution. Ehrlich relates this elective localization to inner causes lying within the tissue. From Ehrlich's work one knows that medicaments are taken up by certain tissues by virtue of their internal peculiarities. Thus, the reticulo-endothelial system has the power to take up toxins. Pharmacologic evidence shows the affinity of drugs for definite parts of the body and the later recovery of these drugs from these parts — the affinity of atropine for the pupillary fibers, aconite for the heart and lead for certain medullary cells. It cannot be denied, therefore, that there are system diseases, and that certain characteristics within the tissue itself determine the localizing vulnerability. This type of vulnerability is spoken of by the Vogts as "Pathoklise."

Toxins may act through the circulation, producing vascular vulnerability in the localization of disease. They may act directly on the tissue but also through circulatory disturbances of a general nature, and these often outlast the immediate effect of the toxins. This is true in carbon monoxide poisoning. Hiller has shown that the pathologic changes in carbon monoxide poisoning are due to the shutting down of the circulation with subsequent necrosis. Thus, the local vulnerability in this disease is due to a local vasomotor disturbance. In morphine poisoning and severe commotio cerebri the disease of the pallidum is also due to a vasomotor factor. Sclerosis of Ammon's horn and pseudolaminar disease of the cortex are likewise due to localized vascular disturbances. In Ammon's horn, the changes are of an ischemic nature. Spielmeyer relates Purkinje cell disease to circulatory disturbances. He agrees with Sträussler and Koskinas who correlate layer-like disappearance of cells in the paretic cortex with circulatory disturbances. Pseudolaminar disappearance of cells has been noted after tying the carotid, in endarteritis tuberculosa and in purulent meningitis. Moreover, it is usually the third layer of the cortex that is affected. What is the basic factor at the bottom of the localization due to local vascular disturbances? Many have been suggested. The blood supply has been implicated in many cases. Hiller relates the involvement of the pallidum in carbon monoxide poisoning to the peculiar blood supply of this region. Uchimura has shown that the involvement of Sommer's sector in the cornu ammonis is due to end-arteries in this region. But Spielmeyer does not believe that this is the entire story in the circulatory localization.

In certain cases, the spinal fluid acts as a localizing factor. Thus the localization of tuberculous meningitis in the spaces above the sulci and in the cisterns is due, says Bohne, to the particularly rich supply of spinal fluid in these regions. This has been corroborated by Spatz in his experiments on sterile meningitis, the process localizing at the base where the fluid supply is richest.

Why is there a predominant involvement of certain cortical layers? And why the particular involvement of the third layer? It is possible to relate this to a physiologic peculiarity of the cells in this area, but this is not the entire story. For though the process seems to be localized often in one layer, it often spreads out over other layers, above and below. The vascular distribution and architecture are factors in the localization. Cerletti has shown how the density and form of the capillary net vary from layer to layer of the cortex. Pfeiffer has shown recently how the cerebral cortex is arranged in layers angio-architecturally. Of particular importance is the mechanical factor of Bielschowsky. In his studies

on hemiplegia with intact pyramidal tracts, Bielschowsky has shown that the vulnerability of the third layer of the cortex is due to the tender make-up of its plasmatic-glious ground substance. Glia fibers are practically lacking in this layer; they offer a certain resistance to mechanical factors. Spielmeyer says, therefore, that one is forced back to the concept of a system disease in explaining the layer vulnerability of the cerebral cortex. He believes, moreover, that Pathoklise limited to a certain definite area has not yet been definitely demonstrated.

ALPERS, Philadelphia.

THE GLIA ARCHITECTONICS OF THE HUMAN CEREBELLUM. A. H. SCHROEDER, J. f. Psychol. u. Neurol. **38**:234, 1929.

The molecular layer, especially its upper third, is abundantly covered with microglia cells and only sparingly with Fañanas' cells. Numerous Bergmann fibers and some macroglia fibers from the granular layer and white substance also run through the upper third. In the middle third the same fibers are seen and just as many microglia cells, more Fañanas' cells than in the upper third and a few oligodendroglia cells. In the lower third the number of glia cells is unusually large, especially between the Purkinje cells. Such large masses of glia cells are not observed anywhere in the central nervous system. Here the microglia is somewhat more abundant than in the middle third; the oligodendroglia may occur in groups, and Fañanas' cells are especially numerous. The principal feature of this layer, however, is the so-called Bergmann zone, a fairly wide zone made up of superimposed rows of Golgi — or epithelial — or Bergmann's cells. The Purkinje cells lie in a veritable nest of glia cells which may occasionally serve them as satellites. The dendrites are often also surrounded by Fañanas' cells, microglia cells, rarely by oligodendroglia cells, and especially frequently by the prolongations of the epithelial cells, which apparently support the dendrites. The entire arrangement of glia cells in this layer leaves no doubt that the macroglia cells are supportive in function. The size of the Purkinje cells, their enormously elongated and thickened dendrites, as well as their direction, location in the outermost layer of the cerebellar cortex and the striking lack of myelinated fibers in this layer, necessitate a strong supportive tissue. It would seem that in pathologic cases the primary object of the glia is to make this supportive function more effective; this is particularly noticeable in Bergmann's fibers, which in the stained preparation appear much thicker and darker, and in the transformation of Fañanas' protoplasmic cells into fiber-forming cells.

In the granular layer all three forms of glia (macroglia, microglia and oligodendroglia) are well represented. The macroglia with its protoplasmic forms occupies an especially prominent position in the middle part of this layer, whereas the transitional forms are more abundant near the molecular zone of the white substance. With their numerous prolongations they apparently form a network which supports the granules. Throughout the entire granular layer one sees the oligodendroglia better distributed than in the molecular layer. The microglia is at least as abundant as in the molecular layer. Pathologic preparations frequently reveal as the first reaction of the macroglia its increase in size and density, leading to the formation of fibrous glia.

The white substance of the cerebellum contains by far the greatest number of glia cells. The macroglia in it is of the fiber-forming type, and it is to this form that the white substance owes its solid structure. In contrast to the number of oligodendroglia in this layer, that of the macroglia and microglia is smaller. Most of the dendrites traverse the large mass of nerve fibers which they surround with their arborizations.

Most of the macroglia cells in the dentate nucleus are of the protoplasmic type, although occasionally Cajal's transitional forms may be seen. On the ganglion cell margin genuine, typical, fiber-forming glia cells are also observed. In the gyrus the macroglia is fairly evenly distributed, appearing in groups of two or three cells. The size of the glial cells is more uniform than that in the white substance.

They are usually star shaped and here and there a form may be seen the dendrites of which originate from two poles. These cells, with the distribution of their dendrites, constitute the architectural peculiarity of the dentate nucleus. The macroglia cells are closely related with the other forms of glia cells, the dendrites of one, two and occasionally of three of these forming a distinct thick covering around the ganglion cells.

More frequently than in any other portion of the cerebellum, the microglia in the dentatum takes the elective stain for oligodendroglia. Oligodendroglia is especially abundant in the dentatum. Usually, several oligodendroglia cells are found around each ganglion cell in the dentatum, but only a few of these are actually satellite cells. The size and number of dendrites correspond almost to those seen in the granular layer. Here and there one may see larger oligodendroglia cells which have long bandlike dendrites.

The Hortega cells are on the average snaller in the dentatum than in other parts of the cerebellum. They are fairly numerous, and after the macroglia constitute most of the satellite cells of the ganglion cells. In the Weigert glia stain one may see longer or shorter wavelike fibers crossing mostly the gray substance of the dentate nucleus.

After concluding the study of the glia in the cerebellum, the author carried out a similar investigation of these three forms of glia in the cerebral cortex (in all areas of the isocortex of Brodmann and in some areas of the allocortex of Rose). From this comparative study, he concludes that: 1. The cerebellar cortex has a uniform glia-architectonics which corresponds to the uniform cyto-architectonics of this cortex. 2. Similarly, a varied but fairly characteristic glia-architectonics in the various areas of the cerebral cortex runs parallel to the varied cyto-architectonics of the same cortex.

KESCHNER, New York.

THE CHARACTERISTICS OF A PSYCHOGENIC HISTORY. T. A. ROSS, J. Neurol. & Psychopath. **8**:287 (April) 1928.

The author's object is to discover whether there are criteria which will enable one to distinguish between physicogenic and psychogenic disease. Careless use of certain words has led to some loose thinking. "Functional" and "organic," for instance, are not opposed words. Every organic disease produces disturbance of function, and it is this disturbance of function of which the patient complains. In a purely psychogenic disease, such as hysterical paralysis, there may be physical changes such as cyanosis of the limb. It is not likely that a neurosis is a minor edition of a psychosis. The difficulties of the neurotic person are the exaggerated difficulties of all normal people. "Neurosis" means a psychogenic disorder, in which there may be physical changes or not; if there are such, they are secondary. The words "mental" and "physical" still cause philosophic difficulties. It is convenient, however, to treat mind as if it existed and mental operations as if they really occurred. It is conceded that psychic traumas do not account for the continuation of symptoms. They are only precipitating events. If certain people are finding psychogeny when it does not exist, others are finding physical disease when it is of no importance. One should study every patient from both the physical and the mental aspect. The presence of a psychogenic history will not assure one that the patient is suffering from a neurosis only, but its absence should make one certain that he is not dealing with such a condition. Hence, one should not diagnose a neurosis in an obscure case unless a psychogenic history is forthcoming.

One must learn to look at patients in longitudinal section. They all have biographies, and one must learn to be a biographer. One must not be content with a history of previous illness or family health but must begin with the early home situation and survey the events of the life. When this habit is acquired it will be found that the psychoneuroses are not so much symptoms of disease as symptoms of a kind of personality. The healthy person tends to find his difficulties lessen as he grows older, for he has acquired more skill in dealing with

them; the neurotic seems to find them more and more difficult, as if what he had acquired was loss of confidence. The normal person is in increasing degree influenced by his victories, the neurotic by his defeats. The former tends to get over his difficulties by himself, the neurotic to rely more and more on something, parents, friends, dogma, systems of health or a physician. In those who have come to rely on physicians, the illness aspect becomes accentuated. Difficulties other than those connected with health tend to disappear from the history. Most of these difficulties begin in childhood. It is often found that childhood was unfortunate in that there was spoiling, anxiety, snubbing, harshness or other mismanagement. Any person giving such a history will require psychotherapy in addition to whatever physiotherapy is indicated. The psychoneurotic does not confess his sins until he comes to trust his physician; rather he tends to blame other people for his troubles.

Three case histories of organic disease and three of psychoses are given to illustrate the lack of the element of increasing disability to meet difficulties and the value of the longitudinal approach. Three histories of neurotic persons are then presented which illustrate how the history enables one to distinguish between the physical and the psychic elements. Such histories take many interviews but require probably no more time than the various procedures of a diagnostic clinic. A diagnostic team should include some one who could be taking a history while laboratory results are awaited. It is nothing new to know that there are definite physical changes in mental states. The question of which are primary remains unanswerable. But it is probable that at present the mental attack is the more likely to succeed and that often when one thinks he is attacking physically he is doing so mentally. The mental is the more difficult form of attack. It often becomes incredibly tedious to both physician and patient; one cannot help wondering whether this may not be one of the reasons for its periodic partial eclipse.

FAVILL, Chicago.

SERVING THE CHILD IN FARGO. PART THREE OF THE FINAL REPORT OF THE FARGO DEMONSTRATION. BULLETIN 9. Commonwealth Fund Division of Publications, 1928.

The central purpose of the five-year child health demonstration in Fargo, N. D., was to show how certain activities designed to better the health of children might be built up as an integral part of a balanced and practicable public health scheme for a small city. In this report the activities are described in sufficient detail to give other cities, which might wish to undertake such a program, the essential facts to work on. The details of the health program are described under three groups dealing with: infants and preschool children, school children and prenatal and postnatal maternity service. In the preschool period the preliminary and most essential step was the establishment of contact by the nurse with families who had young children, in order to interest the parents in adequate health supervision, whether by the family physician or by the demonstration health center. A high degree of cooperative relationship was developed between the demonstration clinic and the medical society, and through the latter and the individual physician the health center undertook no treatment and only examined such children as had no family physician — the purpose of the demonstration being to interest both the parents and the medical profession in developing an adequate supervision of the health of preschool children and to function, not as a foreign body in the community, but as an integral part of the health activities already existing. Of 2,311 preschool children examined by the health centers, 663 were found to have defects sufficiently serious to require medical attention. The most frequent defects in both sexes were dental and nasopharyngeal conditions. Boys showed a large number of genital defects.

Similarly, among the school children the demonstration tried to develop a high degree of coordinated interest between the children, the parents, the teachers and the school nurse. Health topics were discussed by the teacher and class —

the teacher encouraged the children to keep their own health records, and similar records were kept by the parent. There was a daily inspection of hands, teeth, etc., and those children who showed defects were referred to the school nurse. Among school children, dental, nutritional and nasopharyngeal defects were the most common.

The demonstration had three major objectives: (1) to improve the health status of Fargo children; (2) to convince the Fargo public that child health services were worth continuing at local expense, and (3) to make various professional groups more aware of their opportunities to build up and conserve the health of children.

The second objective has been attained in a large measure. It is difficult to measure the exact degree of accomplishment of the first aim, but there is no doubt in the minds of those who have watched Fargo children in the last five years that their health has improved. Infant mortality rates — high in comparison with those of the Registration Area before the demonstration — are now relatively low.

The results of the third aim are still more difficult to measure, but not only has the cooperation between all professional groups been increased, but the citizens of Fargo themselves seem to show a sense of responsibility for the work being carried on, far greater than citizens ordinarily exhibit for the health work of their communities.

This monograph is an interesting record of the manner in which cooperation may be developed among various existing professional groups by an outside agency, the basic plan of which is to work in cooperation with them and utilize their resources to the fullest extent.

PEARSON, Philadelphia.

DELAYED PSYCHOMOTOR AWAKENING, ITS GENESIS AND SIGNIFICANCE. C. ROSENTHAL, Arch. f. Psychiat. **81**:159 (June) 1927.

Delayed psychomotor awakening was first described by Pfister in connection with other disturbances of awakening. By it is understood a period of comparatively short duration, occurring on awakening from sleep, during which the person is unable to move although consciousness is clear. The state is usually accompanied by intense fear reaction. The author reports two cases. Case 1 is that of a woman, aged 35, with a family history of epilepsy and mental disease, and a history of convulsions in early childhood. From the age of 16 she had had attacks of delayed psychomotor awakening which occurred at intervals of from three months to one year. During the attacks she comes out of a deep sleep, knows everything that is going on about her, but cannot move or speak; there is an intense fear reaction. Sometimes the attacks occur two or three times during the same night, and, as a result, she feels tired and listless the next day. At times also, she experiences severe headache. There are also symptoms of disturbances of the vegetative nervous system (intense perspiration, extrasystoles without organic heart disease, etc.).

Case 2 is one of a man, aged 46, whose personal and family history were unimportant. At the age of 37, he received an occipital head injury. This was followed by headaches, dizziness, and, at the age of 44, attacks of jacksonian epilepsy. These attacks are still present but occur at longer intervals and alternate with attacks of delayed psychomotor awakening. The patient awakes from a deep sleep to clear consciousness, but is unable to move or speak. The attacks last from five to ten or even fifteen minutes, and are accompanied by fear reaction.

In these two cases, as well as in most of the cases reported in the literature, there seems to be a relation to epilepsy, either constitutionally or in the patients themselves, and the author believes that the reaction itself is in some way related to the dissociation in epilepsy. He thinks, however, that one must distinguish two different types of the reaction: (1) those occurring rarely and of short duration, without subsequent symptoms; (2) states of dissociated awakening

which recur more or less frequently (sometimes more than once in the same night), last longer, are followed by more or less marked general symptoms (headache, fatigability, etc.), and show an hereditary or a personal relation to epilepsy. These latter he is inclined to look on as epileptic equivalents or some allied states.

The author also discusses the process of awakening in general. One can regard this as a chain of reflex reactions starting in the thalamus, proceeding over the striate body, to the periphery, and back to the thalamus and to the cortex, stimulating these centers into function one after the other. If, for some reason, the original impulses from the thalamus are short circuited directly into the cortex without passing over the striate body and periphery, there will be a dissociation of consciousness and inability to move. The cause of this abnormal reflex process could be sought in the striothalamic apparatus; it may be due to a functional weakness or be an expression of a predisposition to dissociation in the central nervous system in epilepsy. It remains to be seen whether this disturbance in the striothalamic apparatus in cases of epilepsy is due to some irritative organic agent.

MALAMUD, Foxborough, Mass.

RESIDUALS OF A TRUE KORSAKOFF PSYCHOSIS. L. M. ROSENSTEIN and H. G. KLYATZKINA, Collected Papers. Moscow State Neuro-Psychiatric Dispensary **1**:281, 1928.

The authors report the case of a woman, aged 44, who twenty-eight years before had had a true Korsakoff psychosis diagnosed by Korsakoff himself. At the age of 16, in 1900, the patient developed a disease of unknown origin with marked gastro-intestinal symptoms. In the course of the disease she developed severe pains in the hands and feet. After a while it developed into weakness of the upper and lower extremities. At first it was thought that it was a case of Landry's ascending paralysis. Korsakoff was called in. He diagnosed the case as a "polyneuritis with psychic phenomena"—a syndrome which later became known as Korsakoff's disease. Shortly after the onset of the illness, marked impairment of memory was noticed. The patient forgot everything quickly, although she had no difficulty in recognizing her relatives. Within a few months, the patient became better and went abroad to be treated there. At the age of 21, she married and was able to help her husband in a publishing business. She could not remember anything, but she kept extensive notes of what she said and what was said to her and was thus able to take some responsibility.

During the revolution the patient suffered a great deal but stood the strain well. For two years she was employed as a cashier at the race track and was able to do the work well although everybody in contact with her noticed the loss of memory. Her friends tell the most extraordinary stories about her memory.

A neurologic examination showed poor muscle tone and some hypesthesia in both lower extremities. The same was noted in the upper extremities giving a picture of a glove and stocking type of anesthesia. The tendon reflexes were normal. There was ataxia in both extremities, which was more marked on the left; the Romberg sign was positive. No pathologic reflexes were observed.

Mentally she was intelligent, pleasant and good natured. The formal intellectual functions were well preserved with the exception of memory for recent events; there was marked impairment of retentive memory. No delusions or hallucinations were discovered. She showed good insight and judgment. The memory disorder was remarkable. She had lost memory not only for events since her illness but also for childhood and youth. There was complete amnesia for the period of illness and convalescence. The school knowledge was completely gone, and children often reprimanded her for being so terribly ignorant. As a curiosity, she retained knowledge of foreign languages and remembered the principal cities of England, although she forgot the principal cities in her own country. She could not recall when her father and her husband died and com-

pletely forgot about the period of the revolution. Having once read a book, she would usually reread it because she had completely forgotten what she had read previously. She usually went to see the same movie several times but on leaving the movie was never able to recall the plot nor the acting. The formula obtained with the Rossolimo profile test C. was 7,8" 7,2 + 6,7 (59,8 per cent) + 9,3. The personality was well preserved, and the social adaptability of the patient was remarkable. There was occasionally a marked emotional reaction in connection with the inferiority. The author regrets that no pathologic material is available which would throw light on this remarkable condition.

KASANIN, Boston.

EXPERIMENTAL INVESTIGATIONS OF THE SLEEP MECHANISM. G. MARINESCO, O. SAGE and A. KREINDLER, Ztschr. f. d. ges. Neurol. u. Psychiat. **119**:277 (March) 1929.

Three objects were sought in this investigation: (1) the significance of injuries of certain centers for the production of sleep; (2) the effect of anodal and cathodal polarization of these centers, and (3) the effect of certain pharmacologic substances, injected directly into the ventricles, on sleep. In the first group of experiments twenty-three cats were used, and the effect of the injection of calcium and potassium into the periependymal gray matter of the third ventricle was studied. Five of seven cats into which injections of calcium were made gave positive results. An injection of 0.5 mg. of calcium chloride into the lateral wall of the third ventricle caused sleep that lasted from one-half to one hour; an injection of from 2 to 2.5 mg. caused sleep that lasted from three to eight hours. The sleep began three minutes after the injection. The negative results in the series of injections of calcium are explained by the fact that there were large foci of necrosis in the thalamus, causing pain and thus preventing sleep. Animals into which injections of potassium were made showed delayed sleep. The potassium ion, in contrast to the calcium ion, causes excitement which can be observed for from twenty to sixty minutes after the injection. After three or four hours, however, typical sleep develops. Thirteen cats were used for trocar experiments. A cannula was plunged into the brain in the direction of the tuber cinereum, left there for from ten to fifteen seconds and then removed. Six of these experiments gave positive results, showing that a needle injury in the periependymal gray matter of the third ventricle produces sleep. The sleep begins relatively late (from one and three-fourths to three hours after the injury), lasts from three to seven hours, and is variable in its depth. Compared with injection of calcium, the stab wound causes sleep after the interval stated, while injection of calcium causes it after from twenty to forty-five minutes. The interval varies with the size of the dose. The duration of sleep appears to be influenced less by the calcium ion than by stab wounds. A study of the necropsies of the animals used showed without a doubt that there is a relationship between the sleep and the seat of the lesion. To all appearances, foci in the immediate vicinity of the columna descendens fornicis in the periependymal gray matter of the third ventricle produce sleep. The farther the injury from this area, the lighter is the sleep and the more delayed its inception.

Anodal polarization of the lateral walls of the third ventricle in the location indicated produces sleep which begins quickly (from ten to fifteen minutes after the current is applied), is deep, so that the animals are hard to arouse, and lasts for about six hours, the first hour being deep and the last five hours less deep. After cathodal polarization the sleep begins one and one-half hours after the start of polarization, is of medium depth and lasts for three hours. Compared with the result of stab wounds, anodal polarization produces a quicker, deeper and longer sleep.

Injections of ergotamine and calcium chloride into the lateral ventricle produce sleep which begins in from fifty to fifty-five minutes and lasts for from three and one-half to five and one-half hours. Injections of choline and potassium chloride produce sleep in fifty-five minutes, which lasts for two hours.

Necropsies in all the cases indicate that the region which must be injured for the production of sleep is the lateral wall of the third ventricle in the region of the nucleus juxtatrigonalis and the nuclei proprii tuberi.

ALPERS, Philadelphia.

PARTIAL AND GENERALIZED SCLEROSES OF THE CEREBELLUM. WALTER HUBER, J. f. Psychol. u. Neurol. **37**:625, 1929.

Atrophic sclerosis of the cerebellum may appear partly as a local and partly as a generalized process; it involves primarily the cerebellar convolutions and secondarily the white substance of the cerebellum. It is a primary disease of the cerebellar cortex. Macroscopically, the involved areas appear as small shrunken convolutions lying close to each other, appearing on section uniformly white so that the cortex cannot be recognized. The phylogenetically and ontogenetically younger cerebellar hemispheres, the neocerebellum, is frequently the site of predilection for exogenous as well as endogenous lesions and for the atrophies and aplasias involving the cerebellum. In the histologic picture there are no material differences between the generalized and the partial scleroses. Regardless of the etiology, the histology is the same in both; in both the degeneration of the cellular elements and the glial proliferations give rise to a severely shrunken organ. The complete picture of atrophic sclerosis represents an end-result which is stationary, and from which one can readily see that two processes had been at work, of which the later process is the direct result of the first and earlier appearing one.

The completely atrophied portions are connected by means of transitional zones with the healthy tissue, the extent of which gradually diminishes with the progress of the pathologic process. These zones are significant because they indicate the gradual progression of the process to its terminal stage — atrophy. The primary pathologic process consists of a destruction of the ganglion cells, which is followed secondarily by glial proliferation. The cell destruction may appear as a diffuse degeneration involving all layers of the cortex; occasionally, however, it appears more or less in the form of an elective degeneration of one or more systems, especially of the cerebellofugal systems. In the histologic picture these two forms of degeneration (diffuse and systemic) cannot be sharply differentiated from each other; each may be produced by exogenous and endogenous causes and both lead to the same end-result — an isomorphous sclerosis. There is nowhere the slightest evidence of an arrest of development. The entire process is one of genuine cell destruction. Various degrees of cell degeneration and destruction are observed in all layers. The cell degeneration is associated with the formation of broken-down products and a proportionate glial proliferation which eventually leads to sclerosis. All layers of the cerebellar cortex show fiber-forming astrocytes; these are especially abundant in the intermediary zone in the form of Bergmann's glia cells. Glial proliferation begins first in the outermost and innermost layers of the cortex and later invades Bergmann's zone. The superficial fibers extend beyond the original border of the cortex, intertwining themselves with the fibers of the opposite convolutions as well as with the pial fibers leading to a shrinkage of the cortex. As this process goes on Bergmann's layer begins to split up; this is followed by an intertwining of the deeper fibers which eventually leads to a heteromorphous sclerosis.

KESCHNER, New York.

PATHOLOGIC CHANGES IN THE CENTRAL NERVOUS SYSTEM IN EXPERIMENTAL ALCOHOLISM. L. M. DUKHOVNIKOVA and J. A. ROBINSON, Collected papers. Moscow State Neuro-Psychiatric Dispensary **1**:56, 1928.

The literature concerning pathologic changes in chronic alcoholism is contradictory, and one is not sure as to where or what changes take place in the central nervous system. The examination of the brains of chronic alcoholic persons is not always satisfactory because some of the changes encountered may not be due to alcohol. The authors experimented with twenty-one rabbits. From

10 to 50 cc. of 30 per cent alcohol was administered to the rabbits through a pipet until they were definitely drunk. The tests were conducted for a period lasting from several weeks to several months. The total quantity of alcohol consumed was about 300 cc.

The animals were killed by intracardial injection of 20 per cent commercial formaldehyde. Death was instantaneous. Immediately afterward, the internal organs, including the brain and spinal cord, were removed and fixed in 20 per cent commercial formaldehyde. Two or three days afterward, the brain and cord were cut into slices about 2 mm. thick. Frozen sections and celloidin sections were made. The usual stains for the central nervous system were used (Marchi, Weigert, Bielschowsky and Snesareff). Serial sections of the brain and spinal cord were made in about one half of the cases.

The pathologic changes in the central nervous system are similar to those which are found in cases of acute alcoholic intoxication. They are essentially of a regressive character with little glial reaction. There were definite changes in the mitochondria, in the ganglion cells as well as in the glial substance. They consisted in either clumping or complete disappearance of granules. There is nothing specific about these changes as they are found in all other intoxications. The organs most affected by alcohol were the cerebellum, Deiter's nucleus, the red nucleus and the corpora quadrigemina. There was an elective site of cell damage in the second and fifth layers of the cortex. There were also small areas of degeneration in the vicinity of the lateral ventricles.

The authors come to the conclusion that chronic alcoholic intoxication affects whole systems dealing with specific functions. In the first place it picks out the whole group of organs dealing with the functions of position and equilibrium; in the second place, the vegetative centers, and finally the second and fifth layers of the cortex. Alcohol has a selective affinity for the central nervous system. Other organs in the body do not show the same specific changes. Histologic examination shows involvement of the parenchyma. The vessels are affected much less.

Alcohol is not stopped by the hemato-encephalitic barrier. It reaches the brain through the cerebrospinal fluid. This explains the areas of degeneration in the vicinity of the ventricles and the ependyma.

KASANIN, Boston.

ENCEPHALOGRAPHIC STUDIES ON CHRONIC SCHIZOPHRENICS. W. JACOBI and H. WINKLER, Arch. f. Psychiat. **81**:299 (Aug.) 1927.

The studies were made on nineteen cases of dementia praecox. The procedure was as follows: One half hour before the injection the patient was given 1 cc. (0.002 Gm.) of Dilaudid subcutaneously (analgesic). The air injection was done with the patient in a sitting posture. Through a suboccipital puncture, from 60 to 145 cc. of fluid was removed and replaced by a similar amount of air. Except for a moderate headache, and, in some cases, vomiting, there were no untoward sequelae. The temperature usually showed a rise to from 38 to 38.5 C. on the next day. Four roentgenograms were taken: one antero-posterior, one postero-anterior, one left and one right lateral.

Results. In eighteen of nineteen cases there was a definite internal hydrocephalus with dilatation of different parts of the ventricular system. Most of the dilatations were of moderate degree; in a few patients they were slight, but definitely above the physiologic limits. In three of the cases there was also marked external hydrocephalus with narrowing of the convolutions. In six others there was moderate dilatation of the subarachnoid spaces which (compared with numerous encephalograms of normal persons) belonged, without doubt, to the pathologic side. In most of these there was simultaneous narrowing of some of the convolutions. The rather slight dilatation of the subarachnoid spaces in six other cases was not quite as definitely pathologic and could fit into the limits of the normal. Most of the persons studied were of an age when dilatation of the cerebrospinal fluid spaces from senile atrophy of the brain could

not come under consideration. Those of more advanced age (the oldest was 49 years of age) were picked carefully with this in mind.

As in the persons studied the disease process was of rather long standing, the question as to the significance of these observations, whether they were due to a secondary atrophy of the brain or should be regarded as signs of an abnormal constitution, could not be answered definitely. To solve this problem a similar study of a series of "fresh" schizophrenias should be made. This would revive the problems brought up by Bleuler: whether schizophrenia develops on the basis of a special structure of the brain, and whether it can be determined anatomically. The authors believe that if incipient psychoses show similar encephalographic pictures, one would have to look on them as part of such a predisposition. Even if this were not the case, however, the observations still point the way to a renewed study of the possible organic effects of schizophrenic psychoses.

MALAMUD, Foxborough, Mass.

A CASE OF HYDROCEPHALUS IN A DOG. I. N. FILIMINOFF, J. f. Psychol. u. Neurol. **37**:673, 1929.

Filiminoff had an opportunity to observe, in the Institute for Nerve Research in Moscow, a dog whose behavior and reactions during life gave no indication that the animal differed in any way from other normal dogs. Following an unusually large dose of ethylmorphine hydrochloride, which was administered to the dog in the course of some experiments that were undertaken to study his conditioned reflexes, he developed convulsions during which he died. Necropsy revealed a large hydrocephalus, the only portion of the brain remaining intact being the ventral. Behind the presylvian area, the hemisphere appeared in the form of a sac with relatively thin walls. This thinning was especially marked at the bottom of the fissures, but was also prominent in the regions of the convolutions. The centrum ovale had almost entirely disappeared, and the corona radiata was unusually narrow. The convolutions were flattened and the fissures were unusually shallow. The nucleus caudatus, nucleus lenticularis, optic thalamus, internal capsule and mesencephalon were displaced downward and formed the lower wall of the hydrocephalic cavity; these structures themselves, however, showed no essential changes. Noteworthy also was the fact that the cortex, although narrower than normal, was much less involved than the white substance.

According to Filiminoff, hydrocephalus is not at all rare in dogs. The literature also contains several reports of cases in which there was a marked disproportion between the extent of the anatomic changes and the paucity of the clinical symptoms. In this connection the author's case is of special interest. In appearance, conduct and character his dog was normal. As a matter of fact, it was strikingly easy to establish in him the most complicated conditioned reflexes. At the same time, it is hardly conceivable that a dog with such an extensive anatomic defect in the cerebrum could be absolutely normal. It is possible that the deviations from the norm were so fine that they escaped detection in the course of routine observations. The effects of hydrocephalus in dogs as compared with those in man are unusually striking and are of great importance in attempts at comparative interpretations as to the significance of the cerebrum in *Homo sapiens* and of that in lower mammals.

KESCHNER, New York.

THE WHITE BLOOD CELL PICTURE AND THE CAPILLARIES OF THE NAIL GROOVE IN YOUNG MENTAL DEFECTIVES: SECOND COMMUNICATION ON THE SHAPE OF THE CAPILLARIES IN THE FEEBLEMINDED. H. K. KAHLE, Arch. f. Psychiat. **86**:766 (March) 1929.

In this paper the author reports further results in the investigation of the relationship between physical deviations and feeblemindedness. Three hundred and forty-three mentally defective children were examined. The white blood cell counts and differential examinations, as well as the shape of the capillaries of the nail groove, were particularly studied. It was found that the average case showed

an increased number of white blood cells with a relative lymphocytosis and eosinophilia. This was true in most cases, although it was not found in some special groups such as the mongoloid idiots, those with hypothyroidism, the hypoplastic subjects and the dwarfs. The shapes of the capillaries were found to be fairly characteristic and as follows: In the main group of congenital feeblemindedness (in the District of Hanover) the so-called archiocorrected shapes or pseudoneo-capillaries of Hoebfner were found without any tendency toward productive formations. The fact that the shapes of the capillaries are related to certain types of idiocy were made use of in outlining a treatment, especially for that of endocrine deficiencies. Certain small groups of the feebleminded could be differentiated from the main group by the shapes of the capillaries; this is especially so in the case of idiocy due to organic diseases, the athetoid types and those with suprarenal disturbances. The author concludes with a statement that further investigation into the nature of the physical phenomena accompanying mental deviations may be useful in outlining the management of such cases and in determining just what form of training should be instituted in special types. This would also help in attempts at treatment, glandular as well as chemical. The author used tincture of iodine and different glandular preparations. In some cases in which there was a definite indication of the type of endocrine gland involved, that single gland could be used. In other cases, one had to utilize a mixed therapy.

MALAMUD, Iowa City.

ENDOCRINE STUDIES IN DEMENTIA PRAECOX. R. G. HOSKINS and FRANCIS H. SLEEPER, Endocrinology **13**:245 (May-June) 1929.

This study was made up from eighty subjects with dementia praecox and the authors state that at least half of this number gave definite or indicative evidence of endocrine gland deficiency. In fourteen cases the gland at fault was the thyroid and in thirteen the pituitary. In thirteen others the specific gland was not determined. The authors made a rather complete schema for noting the conditions of the patients, and check-ups were made by various members of the staff. Fifty-three of the eighty patients received gland treatment and of these, in at least half of those showing endocrinopathy there resulted significant mental improvement while in the nonendocrine group only five instances of similar improvement resulted from thirty-nine experiments. After the gland treatments five of the patients became well enough to go home and treatment was stopped. Five others, although treatment was being continued at the time the article was written, were almost free from psychotic manifestations. The catatonic group showed the highest incidence of improvement while the paranoid group showed the lowest incidence of improvement; the authors consider that this is probably because of the high incidence of thyroid deficiency in the catatonic group while the pituitary is more at fault in the paranoid type. Thyroid medication proved the most beneficial and mental improvement followed its use in ten of the eleven cases in which the condition was diagnosed as hypothyroid. In eleven nonendocrine cases there was no significant mental improvement following thyroid medication although there was some general physical benefit. The conditions in which pituitary preparations were used showed some improvement in three of the twelve cases. The preparation was given by mouth. In none of the cases in which gonad preparations were used was there any definite evidence of benefit. A few patients were benefited by pluriglandular therapy. They conclude that endocrine deficiency is an important factor in dementia praecox and that in properly selected cases specific endocrine therapy may be of value in the treatment for this condition.

WAGGONER, Ann Arbor, Mich.

CONTRIBUTIONS TO THE ANATOMIC PICTURE OF PARESIS TREATED WITH MALARIA, AND THE PROBLEM OF PIGMENT DEPOSITS IN INOCULATION MALARIA. T. v. LEHOCZKY, Arch. f. Psychiat. **86**:442 (Feb.) 1929.

Twelve cases of clinically diagnosed paresis in patients subjected to fever therapy, who have subsequently died and been examined post mortem, were utilized as the basis of the investigation. In one a histologic examination demonstrated

syphilitic disease of the small vessels of the brain; the others showed a typical paretic process. Ten of the patients had been treated with malaria and two with injections of milk. In the introduction, the author stresses the fact that it is difficult to determine which of the histologic components are the result of the treatment and to differentiate them from those that are part of the paretic picture.

The conclusions reached were: The occurrence of a so-called therapeutic inflammatory process could not be substantiated. Some cases showed pronounced signs of inflammation, but these were of a type that are also found in patients not treated. There is, however, ample evidence that eventually all patients treated in this fashion show a decrease of the inflammatory reaction. This was especially pronounced in seven of the cases examined. The parenchymatous process is slightly, if at all, influenced by the treatment. The author could not substantiate the reports of repair of nerve tissue following such treatments. He considers that the appearances of replacement of lost nerve tissue, as reported by some investigators following malaria treatment, is due to artefact. Bruetsch's observation of special reactions in the reticulo-endothelial system following malaria inoculations could not be substantiated, although Lehoczky found large deposits of malarial pigment in the spleen and liver. There were no such deposits in the central nervous system. Whenever present, however, this pigment could be shown to contain definite quantities of iron.

MALAMUD, Foxborough, Mass.

CHOLESTEROL CONTENT OF CEREBROSPINAL FLUID IN NERVOUS DISEASES. A. S. KULKOV and D. A. SHAMBUROV, Collected Papers. Moscow State Neuro-Psychiatric Dispensary **1**:240, 1928.

Seventy-four patients with various organic diseases of the peripheral and the central nervous systems were studied by the authors. The cholesterol content was determined by a modification of Autenrith's method (Autenrith and Funk: *München. med. Wchnschr.*, 1913). In thirty patients the cholesterol content of the blood as well as of the spinal fluid was determined. Parallel with the cholesterol estimate other biologic and chemical tests were made on the blood and cerebrospinal fluid. The authors accept from 2 to 3 mg. as the normal amount of cholesterol in the spinal fluid.

In syphilitic infections of the central nervous system involving either the parenchyma or the meningovascular apparatus or both there was no increase in the cholesterol content in the cerebrospinal fluid. The blood of these patients showed some diminution in the cholesterol content. There is a definite increase of cholesterol in epidemic meningitis and meningo-encephalitis. This may be due to the presence of cellular elements in the spinal fluid, although, on the other hand, in some xanthochromic fluids the cholesterol content was lower than in clear fluids.

The authors postulate that the increase of cholesterol in meningitis is due to increased permeability of the hemato-encephalitic barrier due to infection. There was also a definite increase of the cholesterol content of the spinal fluid in tumors of the brain and of the spinal cord. It was normal in cases of multiple sclerosis, epilepsy and chronic encephalitis. The authors come to the conclusion that the figures vary so much even in the same condition that one cannot use the cholesterol estimation for practical diagnostic purposes.

KASANIN, Boston.

CENTRAL PAIN. M. LAPINSKY, Arch. f. Psychiat. **81**:197 (June) 1927.

Lapinsky discusses the mechanism of the so-called central pain sensations. Painful sensations in different organs of the body are usually classified into two types: (1) those caused by peripheral pathologic conditions, and called peripherally conditioned pains; (2) those that seem to occur without apparent pathologic conditions in the periphery, and called centrally conditioned pains. The mechanism of the latter has been disputed. Some authors are of the opinion that lesions of certain parts of the central nervous system may cause projections of painful sensations into corresponding peripheral organs. The manner in

which this projection takes place is not clear, although some authors suggest the possibility of an antidromal, that is, a current distally directed from the central nervous system to the periphery. The actual occurrence of such a projection has not been proved.

The author reports eight cases showing this symptom in the course of lesions of the central nervous system; on the basis of these cases, and those reported by other authors, he comes to the conclusion that many so-called central pains may be due to peripherally localizable changes. He is of the opinion that the conditions most frequently responsible for the production of these pains are circulatory disturbances in those regions. They may be caused by irritations of the circulatory centers in the brain (hypothalamus and midbrain); they may also be due to visceral diseases in the neighborhood of the organ affected, and they may be due to misinterpretations of stimuli brought about by disease of the central perception apparatus. Here, however, one has definite stimuli in the periphery which are distorted or changed in the central nervous system, and do not need to postulate projections or antidromal currents.

MALAMUD, Foxborough, Mass.

FULMINATING MENINGOCOCCUS SEPTICEMIA WITHOUT MENINGITIS. RICHARD MIDDLETON and WILLIAM DUANE, Am. J. M. Sc. **177**:648 (May) 1929.

There are two modes of transmission of the meningococcus to the central nervous system: lymphatic and hematogenous; the former has been considered the more frequent, but recently positive blood cultures have often been found before localization of the infection. For this reason vigorous intravenous therapy has been advocated. In some cases the infection does not reach the central nervous system, being only a septicemia; the condition should then be called a meningococcus infection.

There are two forms of septic meningococcus infection: (1) a prolonged febrile state characterized by arthritic symptoms and a mild roseolar eruption; (2) fulminating cases with widespread purpuric blotches. In these cases death may occur very rapidly. A case of the latter type is reported in a youth, aged 18, who died fifteen and one-half hours after the onset of the disease. The spinal fluid was comparatively normal, but the blood count showed 88,550 white cells, many of the cells showing large numbers of intracellular diplococci. A blood culture yielded a virulent strain of meningococci. At autopsy, petechiae were found in nearly all organs, particularly in the suprarenals, which were almost completely destroyed by massive hemorrhages. A sparse growth of meningococci in pure culture was obtained from the surface of the brain.

The authors conclude that meningococcic meningitis is usually preceded by a meningococcus septicemia. They think that vigorous intravenous therapy should always be instituted in case of meningococcus infection.

WAGGONER, Ann Arbor, Mich.

PATHO-ARCHITECTONICS OF THE MICROGYRIC AND PACHYGYRIC CORTEX AND ITS RELATIONS TO THE MORPHOGENESIS OF NORMAL CORTICAL REGIONS. MAX BIELSCHOWSKY AND MAXIMILIAN ROSE, J. f. Psychol. u. Neurol. **38**:42, 1929.

The pachygyric and microgyric cortex bears a close structural resemblance to the primitive four-layered entorhinal cortex, i.e., to the so-called cortex totoparietinus schizoprotoptychos. In pathologic cases a great part of the pallium, which normally shows seven layers, may have an abnormal development of its layers—a development peculiar to the entorhinal cortex. This resemblance is manifested not only by the same number of layers but also by a similarity in the differentiation of the individual layers and of their cellular elements. Thus the cellular layer underneath the lamina zonalis shows, in the entorhinal cortex as well as in the primitive pachygyric and microgyric cortex, a tendency to a nestlike formation of the pyramidal cells. In the entorhinal region, Rose designates this layer as the

"lamina principalis externa." The differentiation of the structural elements in the other layers of the entorhinal cortex is also similar to that in the pachygyric and microgyric cortex. Thus the wide fiber layer (Bielschowsky's third layer) of the lamina dissecans corresponds to the inner layer (Bielschowsky's fourth layer) of the lamina principalis interna. Between the latter and the ependymal layer there is to be found the relatively narrow deep white substance in the pachygyric and microgyric cortex as well as in the cortex schizoprotoptychos.

The results of this method of investigation are of significance because they show that comparative and embryologic studies can support those of teratology.

KESCHNER, New York.

NITROGEN CONTENT OF THE BRAIN FOLLOWING ANESTHESIA. A. REVO, Med.-biol. j. **2**:76, 1928.

Combined morphine-chloroform anesthesia was used on dogs. As far as possible dogs were selected of the same breed, sex, weight and family. In order to remove the blood from the brain the animals were killed by gradual withdrawal of all blood through the femoral artery. Subsequent to this, physiologic solution of sodium chloride was introduced through a cannula into the carotid artery until this solution returning through the femoral artery was colorless. To get norms in reference to the nitrogen content of the brain the same experiments were first done on twenty dogs without anesthesia.

After the vessels of the brain are washed with physiologic solution of sodium chloride and after the brain is completely devoid of blood, one notes that the brain differs in appearance markedly from a normal appearing brain. The gray matter looks grayish yellow while the white matter is a pale shiny white. Samples of the gray matter were obtained by cutting down the outer surface of the cortex, while the white matter was obtained from the lining of the ventricles. Representative samples were examined histologically to be sure that there was no other mixture between the matters. The nitrogen content was determined by the Kjeldahl method and also checked by the author's modification of the Folin-Wu method. The experiments show that as a result of the combined morphine-chloroform anesthesia there is definite increase in the nitrogen content of the gray matter with a partial increase in the white matter.

KASANIN, Boston.

STUDIES OF A HUNTINGTON FAMILY. (A CONTRIBUTION TO THE SYMPTOMATOLOGY OF DIFFERENT STAGES OF HUNTINGTON'S CHOREA.) O. REISCH, Arch. f. Psychiat. **86**:327 (Feb.) 1929.

The author reports a study of five generations of the family of a patient whose case had been reported previously (*Arch. f. Psychiat.* **74**:795, 1926). The purpose was to determine the early symptoms of the disease, before it is definitely established, and also the possibility of the occurrence of abortive syndromes. The cases of definite chorea in this family are reported in detail. Brief summaries are also given of cases which were probably milder, and also of cases in the fifth generation which showed signs especially in the motor system that would point to the possibility of future development of the disease. This is substantiated by the fact that similar symptoms were elicited in the histories of patients in whom the disease has subsequently developed.

Reisch summarizes his observations as follows: Two sisters of the patient previously reported showed symptoms that were essentially those of Huntington's chorea, especially the muscular tension phenomena. These phenomena, although not advanced to the stage of a general rigidity, manifested themselves in intermittent muscle spasms during passive movement. Similar "tension phenomena" were also found in four young members of this family and were accompanied by mild character changes, motor clumsiness and occasional spontaneous movements. Since such symptoms were also observed in the early stages of definite chorea, they may serve as useful early indications of this disease.

MALAMUD, Foxborough, Mass.

THE TREATMENT OF PARESIS BY INOCULATION WITH MALARIA. LEON PRUSSAK, Encéphale **24**:237 (March) 1929.

The treatment for paresis by malaria has shown itself, after an experience of ten years, to be a method immensely superior to any previous one. This method, applied to recent cases of the malady, furnishes 83.8 per cent (according to this author) of satisfactory remissions; in older cases, results are less pronounced. All forms except the "galloping" or foudroyante type are suitable for treatment, with contraindications consisting mainly of badly compensating heart, tuberculosis, persisting thymus and exaggerated obesity.

The chief improvement is in the psychic state; there is little alteration in the neurologic signs. Diminution in serologic observations is variable; usually the greatest improvement is in the lymphocytosis of the cerebrospinal fluid; less in the globulin; still less in the Wassermann reaction, and least of all in the colloidal gold curve. A statistical review of some 5,000 cases reported by forty-three authors yields 29.6 per cent complete remissions and 25.3 per cent incomplete remissions. Factors influencing these results are discussed. Then follow the results of the author in thirty cases (including six of taboparesis) of patients ranging in age from 27 to 54 years, and in all stages of the disease. The majority had had no specific treatment or an inadequate amount. Fifteen of the thirty returned to work; in ten improvement was minimal, and in five (all having advanced cases) no amelioration of symptoms occurred.

ANDERSON, Kansas City, Mo.

THE FATE OF CHILDREN BORN DURING THE EXISTENCE OF A MENTAL OR NERVOUS DISEASE IN THE MOTHER. ALEXANDER PILCZ, Jahrb. f. Psychiat. u. Neurol. **46**:153, 1928.

This is the second contribution to a similar study published by Pilcz in 1924. The results are summarized as follows: Four of thirty-four children born of thirty-two mothers who were suffering from paresis were affected with congenital syphilis, whereas of six children born of tabetic mothers only one showed evidences of congenital syphilis. The mortality during the first year of life in infants born of eclamptic mothers is extraordinarily high; it would seem that after these infants have survived the first year they are, generally speaking, not in great danger of becoming affected with mental or nervous disease. The presence of a mental or a nervous disease in the mother during pregnancy is generally a serious matter for the offspring during the first year of life. Children born of mothers suffering from a mental or a nervous disorder are, up to a certain degree, in danger of being affected with a congenital psychoneurotic defect (feeblemindedness, ethical defect, psychopathy); nevertheless, in the author's material the number of psychoneurotic normal children exceeded that of defective children. The potentiality of these children to become affected psychically later in life is not any greater than is ordinarily the case in children burdened with a psychopathic heredity. It would also appear from this investigation that epilepsy is rarely inherited directly from the mother.

KESCHNER, New York.

EMOTIONAL RELATIVE MONONUCLEOSIS. J. MENKIN, Am. J. Physiol. **85**:489 (July) 1928.

In this study, Menkin utilized a series of normal cats, also cats whose hearts had been denervated, with removal of the stellate ganglia and thoracic and abdominal sympathetic strands. In several animals, likewise, the spleen was resected. Emotional excitement was induced through tying them on a board for from ten to fifteen minutes. Blood smears, stained by the Wright technic, with 200 cells counted, were taken before the beginning of the restraint period, after five minutes of excitement, at the end of the excitement time and at intervals thereafter for from thirty-five minutes to one hour. The data obtained through the procedure indicate that in normal cats the excitement produced by restraint causes a relative mononuclear increase, averaging 13 per cent. This is maintained for about

ten to fifteen minutes following the termination of the excitement period. In sympathectomized subjects, no immediate change in mononuclear count is noted, with the count falling definitely below the starting level from thirty to fifty minutes after the end of the excitement phase. In splenectomized cats, excitement produced a slight rise, averaging 1 per cent as compared to 13 per cent for normal animals and —0.36 per cent for the sympathectomized group. From these results, the author concludes that emotional leukocytosis is mediated through the affective stimulation of the sympathetic system, which in turn induces splenic contractions.

RAPHAEL, Detroit.

CONTRIBUTIONS TO THE PROBLEM OF PERMEABILITY. E. STORRING, Arch. f. Psychiat. **86**:567 (March) 1929.

The author reports his experiences and results with the investigation of the permeability of bromides from the blood into the cerebrospinal fluid by Walter's method. He has used the methods prescribed by Walter, and not the modifications of Hauptmann. The determinations were made with a Burker colorimeter. He used as normal indexes from 290 to 330. Sixty-five persons suffering with schizophrenia and 45 paretic patients were examined, with the following results:

Fifty-six per cent of the patients with paresis showed an increased permeability; 28 per cent had indexes between 290 and 310, and 16 per cent, between 310 and 330. Most of these patients were afterward subjected to malarial treatment; following this treatment a definite change in permeability was found. Of the patients treated, 40 per cent showed an index above 330.

Of the schizophrenic patients, 78.4 per cent showed indexes above 330 (i.e., a decreased permeability); 14 per cent, between 310 and 330, and 4.6 per cent, between 290 and 310; only 3 per cent showed an increased permeability.

These results, therefore, substantiate the observations of Walter and Hauptmann. The author found no definite relationship between the age of the patient and the permeability index.

MALAMUD, Foxborough, Mass.

TREATMENT OF MYXEDEMA IN CHILDREN WITH PREPARATIONS OF THE THYROID GLAND. A. KIESEL, Med.-biol. j. **4**:20, 1928.

The author confines himself to myxedema because it is a disorder of the thyroid gland which is most frequently encountered in children and because it is most familiar to the author. Altogether, 100 children with myxedema were studied and observed. The work began in 1898, and some patients were observed for from several to thirty years. In children from the ages of 5 and 15, the daily dose of thyroid extract should not exceed 0.12 Gm. Larger doses cause symptoms of intoxication with a rapid pulse, flushing of the face, restlessness, epigastric pains, nausea, diarrhea, and occasionally a rise in temperature. In small children, 0.06 Gm. of thyroid is sufficient.

As soon as equilibrium is established with improvement in mental and physical symptoms, half of the previous dose should be given for the rest of the time. The author cites several cases, of girls previously sluggish and underdeveloped, in which administration of thyroid resulted in intellectual improvement as well as the appearance of secondary sexual characteristics. It is much better to keep on giving small doses of thyroid all the time. Periods of rest from the drug were found by the author to be unsatisfactory in the sense of appearance of previous symptoms.

KASANIN, Boston.

EXPERIMENTAL STUDY OF THE EFFECTS OF STIMULATION AND SECTION OF THE VAGAL INNERVATION TO THE BRONCHI AND THEIR POSSIBLE RELATION TO ASTHMA. HARRY T. R. MOUNT, Am. J. M. Sc. **177**:697 (May) 1929.

Following the hypothesis that asthma is essentially a reflex phenomenon and that motor impulses must be carried to the bronchioles by the vagi, the author has made a series of physiologic and histologic observations on a group of dogs,

guinea-pigs and one monkey with regard to the resultant phenomena of various types of vagal stimulation. The types of stimulation used were mechanical and electrical. The tissues of some of the animals were fixed by dropping into formaldehyde immediately; into others, intravascular injections of the fixative were made. In many of the cases changes fairly characteristic of asthma were produced; these changes were not seen in the control animals. The author concludes that his studies establish a physiologic basis for the reflex nature of certain types of asthma.

Since no serious symptoms followed section of the fibers to the bronchi in dogs, it is suggested that in certain intractable cases of asthma section of the bronchial branches of the vagi by means of the extrapleural approach might be considered.

WAGGONER, Ann Arbor, Mich.

THE EFFECT OF LIGATION OF THE COMMON BILE DUCT UPON THE APPEARANCE OF TETANY IN THYROPARATHYROIDECTOMIZED DOGS. J. C. BROUGHER, Am. J. Physiol. **86**:39 (Aug.) 1928.

Brougher's series consisted of six thyroparathyroidectomized dogs; in three the common bile duct had been ligated, while in the other three, this was done and the gallbladder removed. The animals were maintained on a diet of milk, dog biscuit and ground meat, and were carefully observed until death supervened, an average of eight and four-tenths days following operation. On the basis of this study, the author reports that in both groups typical parathyroid tetany did not occur, and when tetanic manifestations did manifest themselves they were definitely slower in their development and were of distinctly lesser severity than is ordinarily the case. In explaining this tetany-inhibiting effect of ligation of the common duct, Brougher suggests the following: (1) lessened excretion of calcium from the body by way of the bile (the liver excretes about 66 per cent as much calcium as is excreted in the urine); (2) increased absorption of calcium from the gut as a result of the absence of alkaline bile acids which precipitate calcium, and (3) depression of the nervous system by the absorbed bile.

RAPHAEL, Detroit.

THE INFLUENCE OF CALCIUM CHLORIDE AND MAGNESIUM CHLORIDE ON MUSCLE TONE IN CASES OF PARKINSONISM. I. I. RUSETZKY, Med.-biol. j. **4**:84, 1928.

Ten patients with parkinsonism as sequelae of epidemic encephalitics were selected for this experiment. Isotonic solutions of calcium chloride and magnesium chloride were prepared and 5 cc. was injected intravenously. The muscle tone was measured by Mangold's sclerometer (described by Mangold in *Arch. f. d. ges. Physiol.*, in 1922). The patients were always sitting in the same position on a high stool with their backs to the wall and feet hanging down. The quadriceps femoris was tested by the sclerometer. Measurements were taken before the injection and for an hour and a half after the injection every ten minutes. The following results were obtained: Calcium chloride definitely increases the muscle tone. Magnesium chloride decreases the muscle tone. A mixture of the two solutions causes only a slight relaxation of the muscle tone. The author suggests experimenting with solutions of magnesium chloride in the treatment for sequelae of epidemic encephalitis.

KASANIN, Boston.

THE BASAL METABOLISM OF SOME BROWNS AND BLACKS IN JAMAICA. M. STEGGERDA and F. G. BENEDICT, Am. J. Physiol. **85**:621 (July) 1928.

The authors carried out careful basal metabolism studies on a series of subjects consisting of forty-two brown Jamaicans, thirty-seven male and five female, and eight black male Jamaicans. They found heat production for the brown males to average only 5.4 per cent less than the Harris-Benedict expectancy for whites of similar age, weight and height. The females averaged 3.4 per cent below the

prediction standard, and the blacks 2 per cent less. These results are rather in contradiction to what casually might be expected, to wit, a lesser metabolic rate in tropical than in temperate climates, in accordance with the earlier belief that in cold environments heat production is greater than in those of higher temperature. That is, from this study, it would seem, as far as the observations may be regarded representative, that climate and diet probably have no pronounced effect on heat production. This work, it should be noted also, is of no little interest as a contribution to racial physiology.

RAPHAEL, Detroit.

THE TREATMENT OF DIABETES INSIPIDUS WITH PITUITARY POSTERIOR LOBE EXTRACT APPLIED INTRANASALLY. JOHN R. CAMPBELL, JR. and H. L. BLUMGART, Am. J. M. Sc. **176**:769 (Dec.) 1928.

Symptoms of polyuria were readily controlled by the use of pituitary extract. There was loss of the "cottony" feeling in the mouth and throat. An abundant flow of saliva was noted, and the skin and mucous membranes assumed more normal characteristics. The patients usually gained in weight. The best method of administering the extract was by means of intranasal pledgets soaked in the solution. When used in this manner the effect lasted much longer than when given subcutaneously. When given by mouth in salol-coated tablets, the extract had no effect. Absorption through the nasal mucous membrane was apparently by the lymphatic route since the application of epinephrine to the nasal mucous membrane (to constrict the blood vessels) before introducing the extract had no influence on the effect. It is important that the pledget be placed high in the nasopharynx, as otherwise the results may not be good.

WAGGONER, Philadelphia.

ON THE MECHANISM OF ISCHEMIC OR ANGIOSPASMODIC PAIN. ALBERT SALMON, Encéphale **23**:290 (April) 1928.

A long review of the literature, as well as extensive personal investigation, leads Salmon to think that angiospasmodic pain can be explained adequately if one gives sufficient consideration to the importance of the venous hyperemia which follows the initial ischemia. This intimate relation of venous hyperemia with pain explains that occurring in intermittent claudication, Raynaud's disease, Volkmann's contracture, the sympathetic syndromes, and also visceral angiospasm, such as angina of the heart or abdomen, and migraine. Evidence for this lies in the fact that Raynaud's disease and related conditions are not painful in the early ischemic period, but become so in the later asphyxial period of venous congestion. Further, cardiac tonics assist some of these illnesses by reducing such venous congestion.

ANDERSON, Kansas City.

ANATOMIC VARIATIONS AND ANOMALIES OF THE SPINE: RELATION TO PROGNOSIS AND LENGTH OF DISABILITY. W. H. BOHART, J. A. M. A. **92**:698 (March 2) 1929. ROUTINE EXAMINATION OF THE SPINE FOR INDUSTRIAL EMPLOYES. B. C. CUSHWAY and R. J. MAIER, ibid., p. 701.

A general summary of 1,000 symptomless spines shows 44 per cent anomalies and anatomic variations, spina bifida occulta being the most common and then sacralized transverse processes. Only those showing lipping or spur formation present lengthened disability or increased tendency to injury. Anomalies of the vertebrae are common and do not necessarily cause painful backs.

CHAMBERS, Syracuse, N. Y.

INCREASED INTRACRANIAL PRESSURE ASSOCIATED WITH SYPHILIS. C. E. LOCKE, JR., Arch. Surg. **18**:1446 (April) 1929.

Locke describes six cases in which signs of intracranial pressure appeared in patients with positive Wassermann reactions. The clinical picture in these cases strongly suggests tumor of the brain. He thinks that while a gumma may be the

cause of the increased intracranial pressure there is evidence to indicate that it may also result from syphilitic leptomeningitis. It is his firm conviction that if signs of intracranial pressure do not promptly subside under antisyphilitic treatments, subtemporal decompression should be performed at once.

GRANT, Philadelphia.

THE EFFECT OF IODIN UPON EXPERIMENTAL HYPERTHYROIDISM IN MAN. DONALD A. CARSON and WILLIAM DOCK, Am. J. M. Sc. **176**:701 (Nov.) 1928.

The effect of compound solution of iodine on experimentally produced hyperthyroidism in man is recorded in four cases. Typical symptoms of hyperthyroidism were obtained by the administration of thyroid extract by mouth. In these four cases, iodine did not alleviate the symptoms which had been caused experimentally. The authors believe that the therapeutic effect of iodine in hyperthyroidism is the result of its action on the thyroid epithelium.

WAGGONER, Philadelphia.

THE TIME ELEMENT IN QUANTITATIVE PERIMETRY. CLIFFORD B. WALKER, Arch. Surg. **18**:1036 (April) 1929.

Walker gives the technical detail of a method which he has found useful in reducing the time needed for quantitative perimetry. He uses a large Bjeurum screen which he thinks will reproduce most accurately the finer details of error in the visual field. By using the technic he describes, more accurate perimetric charts may be made with as much ease and rapidity as the smaller charts at present in use in most clinics. For details of his technic the reader is referred to the original article.

GRANT, Philadelphia.

THE IMPORTANCE OF THE TOXIC ELEMENTS DUE TO NICOTINE AND TO ALCOHOL IN AMBLYOPIA. G. DE VINCENTIIS, Arch. di ottal. **33**:558, 1926; Ann. d'ocul. **165**:621 (Aug.) 1928.

Since de Vincentiis finds toxic amblyopia prevalent in a certain region in Italy where a great deal of contraband tobacco is smoked, he concludes that tobacco is the principal cause of amblyopia due to nicotine and alcohol.

MONOCULAR DIPLOPIA. F. AGNELLO, Arch. di ottal. **33**:561, 1926; Ann. d'ocul. **165**:623 (Aug.) 1928.

Agnello, after having observed monocular diplopia in a girl, aged 20, concluded that it was probably the result of irregular astigmatism due to syphilitic interstitial keratitis.

BERENS, New York.

PROLONGED TREATMENT IN NEUROSYPHILIS. H. C. SOLOMON and M. BERK, Am. J. Syph. **4**:445 (Oct.) 1928.

A series of twenty cases of neurosyphilis is presented, selected on the basis of duration of treatment, showing the favorable results obtained when the usual methods of treatment are prolonged over periods of from three and a half to nine years.

JENKINS, Philadelphia.

OCULAR SYMPTOMS OF TUMOR OF THE BRAIN. MARZIO, Arch. di ottal. **33**:550, 1926; Ann. d'ocul. **165**:619 (Aug.) 1928.

Marzio reports observations in seventy cases of tumor of the brain, in many of which the condition was verified at autopsy or at operation. The frequency of papilledema in tumor of the brain in different situations is commented on, while changes in the visual fields and lesions of the cranial nerves as aids in localization are considered in detail.

BERENS, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, May 7, 1929

LOUIS CASAMAJOR, M.D., *President, in the Chair*

TWO CASES OF A FAMILIAL COMBINED SCLEROSIS RESEMBLING FRIEDREICH'S ATAXIA. DR. JAMES H. HUDDLESON.

These patients were first seen at the Vanderbilt Clinic four months before presentation here. Of the older child, Mi. L., it was said: that her arms and legs had been rather weak, without much change, since the age of 2; that her speech was defective since the age of 4 when she first talked, and that she did not learn rapidly.

History.—The mother had had three pregnancies; the first resulted in a miscarriage; the second and third were with the patients presented here. No history of nervous and mental diseases for three generations could be obtained, but examination of near relatives showed some suggestive signs. The mother's arch was slightly higher than normal, but was not strictly a pes cavus. Her brother and his wife were neurologically normal, but their children (first cousins of the patients) all showed an appreciable degree of pes cavus and a general hyperreflexia, probably within the range of normal. The patient's father was neurologically normal on examination; there was no consanguinity between the parents both of whom were of average intelligence or better.

CASE 1.—The older patient, now aged 11, was born at term by a normal delivery and weighed 7¼ pounds (3.3 Kg.). Teeth appeared at 6 months of age. She sat up at 5 months, talked at 4 years, walked at 9, after the removal of casts which had been applied to correct double congenital dislocation of the hips; she began school at 9 years, and has reached grade 2 B. Crossed eyes were first noticed at the age of 3. She had measles also at 3, and, since then, chickenpox, whooping cough, mumps and scarlet fever; tonsillectomy had been performed and during orthopedic treatment, ether had been given eleven times. There was no history of bladder symptoms or of secondary disorders.

Physical Examination.—The patient was ambulatory and did not appear chronically ill; she cooperated well, except in the more complicated sensory tests. In the cranial nerve distribution, there was alternating convergent strabismus; vision was 20/40 in the right eye, 20/70 in the left; the fundi were normal; there was no nystagmus and no other ocular abnormality. The fifth motor nerve seemed rather weak on both sides. The corneal reflexes were normal, but the fifth sensory nerve distribution was otherwise hyposensitive. Hearing was normal for the usual tests. The palate and tongue were normal, except that there was a slight, fine tremor of the tongue. Muscular strength was generally impaired, the right arm and leg being weaker than the left. The muscles were fairly plump, but flabby, and without demonstrable atrophy or pseudohypertrophy. The deep and superficial reflexes were equally increased on the two sides, without clonus. The Babinski and Hoffman signs were bilaterally positive. The trunk-thigh sign of Babinski was ambiguous.

The gait was slightly ataxic, without reeling, deviation to one side or marked irregularities. The Romberg sign was slightly positive, and the knee-heel tests showed a little incoordination, less of the left leg than of the right. Lordosis of the lumbar spine, and bilateral pes cavus were present. There was slight ataxia of each upper extremity, moderate dysmetria and no past-pointing. In both arms there has been seen an occasional tendency to torsion and a suggestion of athetoid movements of the fingers. Sinistrality was present.

Speech was hesitant; few words were used, and those widely spaced; pronunciation was erratic, with a tendency to slurring.

There was generalized hyposensitiveness to touch and pinprick. Tactile discrimination in the hands was approximately normal (cooperation was limited). The vibratory sense was not demonstrably diminished. The position sense appeared normal for the fingers and for the left foot; it was questionably diminished for the right foot.

The general physical examination showed no abnormalities in the cardiovascular, respiratory or gastro-intestinal systems. The breasts were advanced in development, although menstruation had not been established. The urine and the blood count were normal; Wassermann and Kahn tests of the blood were negative. The mental age was 4 years, 10 months; the intelligence quotient, 32; interpretation, imbecile.

CASE 2.—The younger child, Ma. L., aged 5, was brought to the clinic on account of some mental backwardness and a tendency to scream at night, symptoms that had been noticed for a year or more.

This patient was also born normally at term, and weighed 9 pounds (4.1 Kg.). Teeth appeared at 7 months of age. She sat up at 12 months, talked and walked at 3 years, and began kindergarten at 5. Illnesses were: measles at 6 weeks; tapeworm infestation at 1½ years, which lasted one year and was accompanied by occasional convulsions; whooping cough, diphtheria, and chickenpox; tonsillectomy had been performed.

Physical Examination.—Positive signs in this patient were slight at most. The cranial nerves were normal—clear fundi and no nystagmus—except that the pupillary light reaction seemed a little slowed. No paralysis or paresis was found, though hyperreflexia of all extremities seemed beyond normal limits, with a bilateral Hoffmann but no Babinski sign. The gait was questionably ataxic; standing and walking were done on too wide a base. The finger-to-nose test was executed normally, and the sensorium was normal within the limits of testing. This patient's speech was much freer than her sister's, but exhibited a little slurring at times. The results of the general physical examination, urinalysis and the blood morphology and serology were normal. The mental age was 3 years, 8 months; intelligence quotient, 70; interpretation, borderline.

Summary of Cases.—A mild, ataxic quadriplegia, commencing at the age of 2 or sooner, accompanied by slurring speech and marked mental retardation and without a history of early trauma or severe infection, was seen in a girl, aged 11, of intelligent Russian Jewish parentage, whose sister, aged 5, has a similar though abortive symptomatology, and whose three first cousins show pes cavus without other neurologic signs.

DISCUSSION

DR. J. W. STEPHENSON: I do not see the resemblance between the cases presented and Friedreich's ataxia. The latter is a combined system disease, in that two or more systems may be involved, the characteristics being involvement of the posterior columns and pyramidal tracts. These cases do not show any alteration in vibratory sense nor is there any postural defect. The question of the existence of pes cavus is open to doubt. When the children walk, the deformity looks more like flat feet. The chief feature of the cases as presented is the involvement of the pyramidal tract, which I believe to be of familial type.

DR. LOUIS CASAMAJOR: I feel somewhat the same as Dr. Stephenson concerning these patients; I do not think that anything has been proved by this presentation, least of all that the children are suffering from Friedreich's ataxia. They did not behave well, and that was an added difficulty with which Dr. Huddleson had to cope in presenting the material. One is under a great disadvantage with such refractory patients, but there does not seem to me to be any condition to diagnose, except the possibility of involvement of the pyramidal tract.

DR. HUDDLESON: These cases were not presented as Friedreich's ataxia, but rather as cases similar to that condition. Such cases were described in 1892 by Sanger Brown, and later grouped by Oppenheim with others not corresponding to Friedreich's or Marie's ataxia, but with similar familial tendencies.

A Theory of the Genesis of Tremor in Lenticular Disease. Dr. Samuel T. Orton.

It is held important that the character of a lesion as well as its location be borne in mind in explanation of the symptoms related to it. In the lenticular diseases, especially in Wilson's disease and pseudosclerosis, the lesion is characteristically a destructive degeneration of cells with little of the glia reaction associated with the discharging or irritative type of lesion. One of the symptoms of this destruction is the appearance of tremor, which at first sight looks like a positive or discharge phenomenon. In view of the rather purely destructive character of the lesion, one may inquire as to how such a positive symptom might arise.

The lower motor neuron is known to have a natural rhythm of response in reflex movements of from about 7.5 to 12 per second which corresponds fairly well with the tremor rate, and the tremor has been assumed to be a lower motor response. The neuromuscular mechanism, exclusive of the anterior horn cell—i. e., the nerve-muscle preparation—has been shown experimentally to have an unlimited capacity to respond in harmony with the stimuli applied. When the anterior horn cell is also included the situation is more complex, but the motor reflex arcs can respond within a wide range of rhythms to the control of the pyramidal tract. Intrinsic rhythms of discharge seem to be a universal characteristic of nerve action, and one may assume that the pyramidal system also has a fixed rhythm of response; there is some evidence to suggest that this also is at the rate of from 8 to 10 per second. The lenticular system forms a sort of by-pass from the pyramidal tract to the anterior horn cell by way of the pyramidal collaterals to the lenticular nuclei and their pathway via the red nucleus and rubrospinal tract. The pyramidal tract, to establish its dominant control over the anterior horn cell, probably maintains a continuous flow of subliminal stimuli. The suggestion is made that part of the function of the lenticular by-pass is to prevent the expression of this continuous flow as a tremor, and that destruction of any part of this adjuvant motor pathway allows the pyramidal tract rhythm to be implanted on the anterior horn cell, resulting in tremor. This view would be in accord with the occurrence of tremor in conditions characterized by destructive rather than discharging lesions.

DISCUSSION

Dr. Frederick Tilney: To me, the question of tremor has been and still is most perplexing and difficult. In listening to Dr. Orton I have been impressed by several of the suggestions which he offers in the explanation of the so-called lenticular type of tremor. The relation which the striate body bears to the organization of somatic motion is still a matter of debate, but this thought of Dr. Orton, that the tremor is really a cortical one with definite pyramidal expression, has especial interest for me. According to this view, cortical areas are permitted to express themselves in the musculature in such a way that the ordinary rhythm of from 8 to 10 pyramidal flow emerges through the lower motor neuron innervation in a definite tremor rhythm, which may disappear as soon as it is inhibited or controlled by a special subcortical mechanism. This seems to me a rather new interpretation and an appealing one, particularly in view of the fact that Dr. Orton has offered as evidence the modifying or inhibiting conditions of sleep and anesthesia and of hemiplegia occurring intercurrently in the course of lenticular disease. Whether this explanation of a cortically determined tremor is entirely satisfactory to all of the facts as we known them, must, I think, still remain a matter for further study. In my own studies of tremors of this type, I have been impressed by the fact that a large degree of cortical control does enter into the picture of striate tremor. In the production of this tremor the two great groups of muscles cooperating in the production of it, that is, the antagonists and the agonists, show a definite cocontraction rhythm. That is true not only of Wilson's disease, but also in most cases of paralysis agitans, for which reason I have long believed that the cortex cannot be overlooked in the explanation of lenticular tremors in general. All steadiness of motion, all motion which is well coordinated and well balanced,

depends not on any single system, but rather on the combination of systems which enter into the production of motion. For this reason Dr. Orton's suggestion is particularly appealing to my mind as it gives a clear explanation of the combination between the striata and the cortex, which I think has not hitherto been fully appreciated. I have been particularly impressed in the study of tremor with the difference between the disorder of motion which occurs in voluntarily produced movement, as in multiple sclerosis, and that type which we now choose to call "lenticular." The difference is so striking that when compared in kymographic records one can easily differentiate "rhyhtmic flow" in the tremor of paralysis agitans and that pronounced arrhythmia in the grosser disorders of cerebellar defects. It is my belief that in this latter type one sees the effects of cerebellar disease in contrast to the defects which occur in lenticular tremor as illustrating cortical or corticostriate disturbances.

Whether this disturbance is a defect or an irritative phenomenon, I am not prepared to say. I should be inclined to believe that it belongs more distinctly to the defect rather than to the irritative group, for certainly the pathologic pictures, as shown in Wilson's disease and in paralysis agitans, would lead to the belief that the defect exists somewhere in the corticostriate or the rubrospinal or the striatorubral connections when tremors of this type are present. It seems to me that any interpretation in this difficult field, any theory which projects trial suppositions even far ahead of the facts, is a fair means by which one may attempt to approach the truth. For that reason I am grateful for Dr. Orton's explanation.

DR. WALTER M. KRAUS: Dr. Orton has done a service in that he has given a theoretical basis for something which is, at least to some degree, clinically established. It has been the opinion of many, from postmortem evidence, that the neostriatum is related to tremor. Experimentally, I do not believe that there is any evidence which would indicate that removal of the neostriatum will permit tremor to occur, and I should like to recall Magnus' statement in regard to athetosis and chorea, in which he emphasized that removal of the basal ganglia in lower animals never produces these curious pictures which one sees in man. Thus, for the present one must depend on clinical observation and pathologic proof of one's suppositions in the living patient to increase the knowledge of the genesis of tremor.

As one thinks of the circuits of the nervous system and of the various nuclear masses which may be concerned in the production of tremor, one sees why the neostriatum in particular may play a part. Though one knows that the neothalamus is not solely concerned with the sensations derived from somatic receptors —for example, the anterior nucleus of the thalamus is concerned with smell—yet the neothalamus is concerned with those sensations such as visual, auditory and proprioceptive, which play a guiding rôle in movement. From the neothalamus, these impulses are transmitted to the neostriatum. Therefore, it would be logical to conclude that a break in the circuit due to disease in the neostriatum will permit the occurrence of an abnormality of movement.

What I said at the beginning remains as the delightful part of the paper. One has the feeling that conclusions derived from a group of clinical and pathologic observations are confirmed by a method of reasoning based on neurophysiology. Naturally, one thinks of a great many other forms of tremor. One thinks of rubral tremor. In those cases the influx from the cerebellum is also lost and the tremor is of necessity of different character. On the other hand, one thinks of toxic diseases, such as exophtalmic goiter, in which there is no demonstrable lesion of any of these nuclei. One is not at all certain that the thyroid gland does have its effect as much on the neostriatum as on the neocortex. It is difficult to adjust these conditions to the theory.

However, I feel that Dr. Orton's paper has given a stimulating approach to the problem. It furnishes a method of thinking about it, and after all, that is the most important part of any such problem. Probably the clinical application of the theory which he has advanced will be found to have flaws in it, but I think one will find that the major premises are correct.

DR. SAMUEL BROCK: May I point out a seeming objection in Dr. Orton's excellent thesis? This is embodied in S. A. K. Wilson's discussion of tremor ("Modern Problems in Neurology"), in which he stresses the part played by cerebellomesencephalic and diencephalic mechanisms, the integrity of which inhibits the tremor's appearance. He points to a rather important fact which Sir James Parkinson had noted in his monograph on paralysis agitans; viz., a patient afflicted with this disease suffered a hemiplegia, and the tremor disappeared on the paralyzed side for a period of weeks; later the tremor reappeared, although the signs of a disease of the pyramidal tract were still evident. Wilson, too, describes similar cases. In some of them, the tremor reappears in the presence of signs of involvement of the pyramidal tract when the period of shock is over. It is not altogether easy to harmonize this fact with Dr. Orton's theory, but if I may take Wilson's view of it, he comes close to an implication contained in Dr. Orton's hypothesis; i. e., tremor is a very low order of involuntary movement, much lower than chorea or athetosis. In chorea and athetosis, a greater variety of movement is seen; especially in the former, a variability of pattern may be easily discerned. But in tremor, the movement is strikingly uniform in respect of pattern, amplitude and tempo. Wilson refers tremor to a peculiar "release" of the anterior horn cells. He believes that the reason why the hemiplegic insult removes the tremor in paralysis agitans is one of diaschisis. In the period of shock, the pyramidal tract insult "jars" the synaptic relations of the anterior horn cell and disturbs its functions. The tremor disappears. Later, as the functions of the anterior horn cell are resumed, the tremor reappears. Viewed in the light of Wilson's conceptions, tremor becomes a low form of involuntary movement, mediated by the activity of the anterior horn cell, released by disease from the control of higher tremor—inhibiting mechanisms situated in the diencephalic and cerebellomesencephalic regions. This point of view also brings up puzzling questions. Why should anterior horn cells so released possess this rhythmic, seemingly purposeless activity? Why is such an elaborate neural mechanism necessary to prevent or to inhibit the appearance of tremor? As yet much too little is known in this perplexing realm of disturbed neural function.

DR. J. W. STEPHENSON: I had occasion recently to discuss with Dr. Orton the behavior of tremor in a case of paralysis agitans which I have had under observation for several months, and subsequent developments in this case have disproved the theory as advanced by Dr. Orton. This patient has an advanced case of paralysis agitans and for three years has been practically helpless. Five months ago, he developed a left hemiplegia. With the development of the hemiplegia, the tremor stopped. Until three weeks ago, function had been restored to the point where the patient could put a cracker into his mouth, put his hand to the top of his head, pull on and push off bedclothing. He had been unable to make any of these movements for at least three years. With the restoration of this function, there has been no return of the tremor. Three weeks ago, he had another stroke involving the left side. Today he can slightly flex the fingers and pronate the forearm, but there is no tremor. As I understand Dr. Orton's theory, the pyramidal system possesses certain inherent rhythmic tremors which are controlled by the lenticular mechanism. If this is true, I ask Dr. Orton why with the return of function in this case there has not been a return of tremor? In Wilson's recent treatise on athetosis and tremors, the statement is made that the tremor in paralysis agitans disappears after development of hemiplegia, but one implies from his remarks that the tremor returns on improvement of the hemiplegia. I gather that Wilson thinks the tremor stops on account of indirect involvement of the anterior horn system, and if my inference is correct, the restoration of function in my case also disproves his theory. From the study of this case I have concluded that neither the lenticular structures, the cerebellum or the mesencephalon is, per se, responsible for the tremor of paralysis agitans. Time will not permit me to explain my views, but suffice it to say that they can explain the tremors observed in emotional states.

DR. E. D. FRIEDMAN: The impulse of voluntary effort presumably travels along the pyramidal tract. May I, therefore, ask how Dr. Orton would explain the ability of the patient with Parkinson's disease to control the tremor by such an effort?

DR. SMITH ELY JELLIFFE: I hesitate to arise because either through deafness peripherally or stupidity centrally what Dr. Orton has said is as clear as mud to me. In the first place, I found nothing new or inclusive enough in the paper of Dr. Orton to entitle it to the dignity of a theory. He has only repeated certain facts concerning the pyramidal system which are fairly well known. Dr. Brock has already called attention to Wilson's discussion of the effects of hemiplegia on certain types of tremor, and one knows that Parkinson, a century ago, described cases of paralysis agitans with intercurrent hemiplegias in which the tremor was lost for a while, and when the patient recovered his power over the pyramidal tract system the tremor did not reappear; in other cases the tremor did reappear. I tried to understand what Dr. Orton was saying when discussing the cholesterols and other types of toxic products. As I rather hazily gathered, such experiments tended to show that irritative phenomena resulted from these toxic products, either directly or through tissue alterations. What relation these experiments had to the problem of tremors I did not gather. I wish to call attention to some experiments which de Jong has been making on tremor with bulbocapnine. How would Dr. Orton explain the results of this alkaloid on the tremors of Wilson's disease and cases of encephalitis on the basis of the sketchy hypothesis he has presented?

I am much interested in that aspect of Dr. Orton's paper concerning the emotional relationship to the production of the tremor. Some of you will remember some of the slides which I showed here a year or so ago from a respiratory encephalitic case in which, while writing out a dream in which the patient "took a stick and threatened to strike the policeman," a typical paralysis agitans tremor appeared in the words "threatening to strike." Herein it may seem that a cortical situation is of significance. Whether it is cortical, however, I have my serious doubts. I believe it is possible to get such a result from diencephalic level action. I thoroughly agree with Wilson's more or less general statement, which Dr. Brock has brought out, concerning the primitive nature of certain tremors. Wilson insists on the subpallial significance of tremors. I wish Dr. Orton would clarify certain of these situations, since, so far as I have heard him, he has discussed only certain simple efferent factors in a highly complex dynamic situation. Take the ordinary phenomenon in paralysis agitans of closing the fist tight, when the tremor subsides, and then it is resumed, or the paralysis agitans tremor when the arm is put at rest. Here the tambour records a definite modification of the tremor; or again, when the individual is put in water, and gravity stimuli are taken away from the muscles, the whole situation is modified. The limb in water loses all of its tremor. Certainly the pyramidal tract is still operating whether the patient is standing inside or outside of water, but something has taken place which I did not hear explained along the lines which Dr. Orton has taken. I think Dr. Tilney was entirely too diplomatic to the reader of the paper when while first emphasizing the fact of the extreme complexity of the tremor situation he thought Dr. Orton's "theory" was valuable. The fragment of an hypothesis that Dr. Orton has offered is not worthy of the name of a theory; it is entirely too simple, and to my mind, at least, it hardly explains any of the complexities of the problem.

DR. SAMUEL T. ORTON (in closing): So many questions have come up that it is obviously impossible to answer them. When tremor ceases after hemiplegia it is clear that one is dealing with a different situation entirely, and until such cases have been adequately studied anatomically it is impossible to say where the lesion was, or to interpret it further. I did not intend, in giving this simple offering to put it on the basis of a revolutionary theory, but I believe in bringing together certain facts which may suggest further investigation.

I feel definitely that study of this problem in animals will not be of help because one is dealing with a mechanism which is very different in the human and animal brain, and it is impossible to transfer immediately the results of animal experiments

to man. The dominance of the pyramidal tract is markedly greater in man than in any of the animals. Further, it is impossible to make a truly selective lesion of any part of the central nervous system without damaging other parts. Apparently the only hope one may have of that approach is through the use of a selective toxin. Many toxins are highly selective for the central nervous system. If by means of a chemical approach one could develop a neurotoxin of a selective type, comparable to the suggestion that Wilson made in his original article of the selective character of the bile-staining of the lenticular cells in icterus neonatorum, one might produce a lesion in the animal which could be used for comparison, but the operative approach seems to be hopeless.

The question which Dr. Brock and Dr. Stephenson brought up is unanswerable. I do not understand the situation there at all. Obviously, tremor may be derived from the anterior horn cell. As I pointed out, the anterior horn cell is known to have a rhythm of response of from 7.5 to 12, and tremor may be purely a lower neuron response. Dr. Stephenson's case seems to be particularly striking with the double hemiplegia and subsequent loss of tremor but with recovery of movement. I am frank to say I see no definite explanation of that either from the basis of a low level or a high level origin of tremor.

With regard to the voluntary control of tremor, I have puzzled much over that. One of my cases a number of years ago proved to be one of pseudosclerosis, and I was misled decidedly in the diagnosis because of a relatively high degree of voluntary control. The patient was unable to walk and had a great deal of spasticity and tremor, but under the stimulus of following somebody could walk across the ward. On that evidence I made a diagnosis of hysteria, but at autopsy there was a hobnailed liver with diseased lenticular nuclei. Microscopically, the case proved to be one of typical pseudosclerosis. It is possible that under great volitional effort the tremor is in a certain degree controlled because of the extra charge of the pyramidal tract.

I do not take Dr. Jelliffe's remarks to heart, since I have heard him discuss many papers by many authors. As far as rest and support are concerned, it is obvious that the pyramidal tract plays a part aside from that in volitional movement. One is taking off the limb a large part of this pyramidal tract influence when the support is brought to bear. The pyramidal tract is not operative only during volitional motion, but it is constantly operative on the anterior horn cells. The relief by support in water or by resting on a table will necessarily reduce this type of control.

FURTHER LIGHT ON THE TRANSMISSION OF PAIN AND TEMPERATURE WITHIN THE SPINAL CORD: HUMAN CHORDOTOMY TO ABOLISH PAIN SENSE WITHOUT DESTROYING TEMPERATURE SENSE. DR. BYRON STOOKEY.

It has generally been taught that the transmission of pain and temperature is through the spinal thalamic tract; however, there have been numerous investigators who have doubted that pain and temperature were carried in the spinal cord in this tract. Dr. Strong has called attention to the fact that perhaps pain and temperature may not be carried in this one tract, first described by Gowers. However, most of the knowledge on which this doubt was based has been drawn from gross pathology and not from discrete lesions. It is well known that any tumor of the brain or spinal cord which is far enough advanced to give signs to indicate the presence of such a tumor produces rather massive distortions of the cord, and as the spinal thalamic tract is small, perhaps 5 mm. in thickness, any distortion sufficient to give rise to massive cord signs could hardly be applied to the finer interpretation of the transmission of pain and temperature. Likewise, gross pathologic lesions such as are found in syringomyelia are not sufficiently discrete to warrant one to draw deductions concerning whether or not pain and temperature are carried in this tract. Gowers was the first to call attention to the fact that probably pain and temperature were carried in the anterior lateral columns. Van der Heuve, in 1893, was the first to feel sure this was true, and Spiller, in 1925, made the statement that pain and temperature were carried in this vicinity and in the spinal thalamic

tract. In view of this situation, attempts were made to abolish the pain and temperature in gastric crises and in other painful attacks in which no other form of relief could be obtained. The operation was first done by Schiller, of Germany, in 1908, and later by Krauder and by Nonne; it was first performed in Philadelphia by Martin, in 1911, and the second case was done by Beer in New York. Both of these were anterior lateral sections, and in both pain and temperature were abolished. Our operation came into vogue in 1920, when Fraser collected a series of six cases; in 1923, he collected eight cases, and in the literature up to the present time there are over fifty cases. Fraser developed the operative procedure and recommended the use of a sickle-shaped knife, so that when this is inserted into the cord at a certain point and is brought out it will automatically cut the spinal thalamic tract. As the spinal thalamic tract is much more superficial than one is led to believe from the use of this knife and the operative procedure advocated by Fraser, when the opportunity to do a unilateral chordotomy presented itself in a case of Dr. Foster Kennedy's, instead of using a sickle-shaped knife, the direction of which one cannot control, with the visualization of the spinal thalamic tract in mind, I did a free section with a fine razor blade no. 11 Barr-Parker knife into the spinal cord. This was not entirely successful, and the next morning, a slightly deeper section was made. On examination, this patient could distinguish a pinpoint as a point, but it did not carry the affect of pain; temperature sense was retained. This procedure has been done in six cases, in each of which the temperature sense was retained and the pain sense abolished. The technic of this operation differs little, if at all, from that of those neurologic surgeons who have done this procedure. The only points to be emphasized are that instead of doing it with the sickle-shaped knife it is done with the knife described, and that the cut is made into that point which is approximately opposite the lateral part of the ventral horn; if the incision is carried in there and not out to the ventral portion, the temperature sense is not lost.

There has been a great deal of discussion about the lamination of the spinal thalamic tracts. I believe that there is some sort of lamination, but I cannot prove it in this series of discrete lesions, in that sensation for pain will be completely abolished for the fifth, fourth, third, second and first sacral segment and for the fifth lumbar segment; it will be present in the fourth lumbar segment; then the third and second lumbar segments and on up to the level of the section will have lost sensation for pain; so in, the middle of the area that presumably is laminated in the spinal cord, a segment is omitted.

There has also been a great deal of discussion as to whether or not visceral pain sense and deep pain sense travel in the same route as superficial pain sense. One case is cited which proves that visceral pain sense is carried in the same tract as superficial pain.

There are other conditions besides inoperable tumors in which the surgeon is compelled to find relief for intractable pain, the cause of which may not be known.

I feel that I know where the pain tract is, but I do not feel I have sufficient evidence as to where the temperature tract is. I think it is either more ventral or more mesial, but I do not know which.

Another point that is important in doing a bilateral chordotomy is to make the cut in one segment and to skip two segments before making the cut on the opposite side. This is a factor of safety, and it avoids the danger of throwing out the entire segment due to its blood supply.

In no case have there been any pyramidal tract signs or paralyses. In one case, there was temporary incontinence.

It is a tremendous advantage to these patients to destroy the pain fibers without destroying the temperature fibers. They can get into a hot bath without burning themselves.

DISCUSSION

DR. FOSTER KENNEDY: Dr. Stookey, just at the end of his paper, spoke of trigeminal neuralgia and the subtotal operation for its relief. In the field of trigeminal neuralgia Dr. Stookey has shown himself to be exceedingly expert,

and I think that he has added materially to the operation of chordotomy by his contribution. The operation on the patient, of whom Dr. Stookey spoke as his first patient, so far as I know, was a complete success. He said parenthetically that for various reasons it was not a success, but that was an obscure statement to me; I do not know what he meant by it. Perhaps he meant that he did not consider it a success because in his first effort to cut the spinal thalamic tract he went short of his objective; to our disgust, on the evening of the operation and the next morning, we discovered that no important loss of sensation had been attained. Perhaps this was the matter in which he thought it was not a success. However, a surreptitious operation was done on the next morning under the guise of a dressing, and the operation was carried out successfully. We were much astonished to find that temperature was retained and pain, as an emotional quality, lost. I have been kicking myself for some time because of my astonishment. I think I was stupid in that I have been seeing cases of segmental syringomyelia for many years in which in certain segments pain was lost and in certain segments temperature was lost, as quite discrete deficiencies. The fact that syringomyelia is capable of abolishing pain and retaining temperature, and vice versa, ought to have made it clear that temperature and pain do not run precisely in the same bundles; that we should have been suprised at the result of Dr. Stookey's operation was a lamentable failure in reasoning.

I think another contribution that Dr. Stookey has made is his suggestion of the wisdom of jogging the operation of bilateral chordotomy by doing the section at different levels. I am sure that this is a wise procedure, when bilateral chordotomy is called for.

DR. ALFRED S. TAYLOR: Dr. Stookey has presented the subject of the surgical technic of chordotomy so well that there is not much left to say; therefore one can only agree with him, and perhaps make one additional statement, that no form of fixed instrument, curved or straight, will do the same definite thing to every cord. I think those of us who have seen cords frequently will appreciate the fact that they vary in size the same as individuals vary in size, and therefore it would seem to be a bad principle and bad practice to have the same fixed instrument for operation on every spinal cord. The semicircular steel hook would pick up too much in a small cord and possibly too little in a large cord. If my cord was to be divided, I should choose Dr. Stookey in the first place to do it, and I should like him to visualize the portion of the cord to be sectioned, and then to take his knife and cut free hand.

DR. OLIVER S. STRONG (by invitation): With all due deference to tremors, I think it may be said that sensation presents an even greater number of problems which await solutions. All along the line, so to speak, are interrogation points, both anatomic and physiologic, which must receive answers before the mechanism of sensation becomes clear. In this field, too, the limitations of the experimental method are especially great. Owing to their lack of language reactions, as well as to other differences, animals are not available for tests of other than the grosser forms of sensibility, and the interpretations of such reactions as are obtained are often open to doubt. Doubtless, more might be obtained by the tedious methods of isolated conditioned reflexes, but such results would not compare in variety or accuracy with those obtained from human beings, although even in testing the latter there are many pitfalls. This greater availability of human beings, by the way, is one of the reasons for considering such papers as the one under discussion of the greatest value.

In a discussion of problems relating to sensation one is met at the outset by the fact that the receptors themselves present important unsolved questions, especially those involved in the forms of sensibility under present discussion. While the receptors for the sensibilities of position and movement are to some extent at least fairly well established, and also those for certain forms of touch, the receptors for pain and still more especially those for temperature present many obscure points. Are "diffuse" nerve terminations the only nociceptors? What is their relation to the pain spots, and how is their apparent greater accessibility to stimuli as com-

pared with the encapsulated endings reconcilable with the higher threshold of the former? Does the answer to the last question lie in the not improbable assumption that the diffuse endings are of finer fibers, both myelinated and amyelinated, and consequently of slower transmission? Fine fiber terminations are also present in many encapsulated receptors along with the regular coarse fiber terminations belonging more properly to them. Are the former pain fibers or branches of pain fibers, and does their presence here account for the intimate blend of, for example, a tactile and a disagreeable component in ordinary pain to pin prick? The same considerations apply to deep pain, not only pressure pain, but also pain from over-extension or overflexion of joints—a form of pain not sufficiently tested ordinarily by neurologists—in spite of its extensive use by children and wrestlers. In general, how far do we try to test by receptors in sensory examinations, e. g., take cognizance of discrete pain spots in pin prick, of the difference between stroking and isolated applications of a pin, of hair sensibility, and that not only by stroking but testing localization and two point discrimination by stimulation of individual hairs? In regard to interpretation, in testing by pricking is "sharp" to be interpreted as a discriminative or a pain reaction? In testing position and movement sensibility, how far is the error of unequal pressure avoided and also a distinction made between an abrupt and a gradual change of position, the latter probably having a higher threshold?

There arises also the question, raised by Boeke, Foerster, and others, of a peripheral, intra-epithelial, neurofibrillar and periterminal net of such a character that stimulation of the peripheral end of a cut nerve may pass "antidromically" to the net, through it, and loop back through another nerve, thus still giving rise by this roundabout route to sensation. One also encounters the important questions, both histologic and physiologic, relating to vascular sensibility, especially pain, and its relation to the autonomic system. Then too, there are the numerous and extremely important questions arising in connection with visceral pain and its receptors, not to mention other forms of visceral sensibility and the question as to their respective assignments to the sympathetic and parasympathetic divisions of the autonomic system. I have always thought, following both anatomic arrangement and the broad lines laid down especially by Cannon, that the part played by the sympathetic in visceral affairs is that of intervention in the interest of the external emergency situation and consequently a stoppage of the purely visceral activities. Included in this sphere, however, are the nociceptive visceral as well as external stimuli. On the other hand, the parasypmathetic regulates the specific functions of the various viscera under the influence of stimuli coming from them; this applies also to the eye (sphincter iridis by retinal stimuli, dilator by sympathetic). In this case the specific organic sensations (e. g., hunger, thirst, etc.) should be mediated by the parasympathetic (e. g., vagus).

The character of the thermoceptors appears to me even more doubtful than that of the nociceptors and the assignment of cold and hot spots to certain encapsulated receptors arbitrary. The intimate relation between thermal stimuli and the vasomotor system should be taken into account here.

The establishment of the various receptors forming the starting points of the various kinds of sensibility is the foundation stone of a scientific knowledge of sensation. One of the prerequisites of this is a careful census of all the receptors of the human body, both as to number and distribution. Such a laborious task might well be the major work of a research institution.

It is evident from what has been said and also from recent physiologic work on conduction along nerves, that the more exact constitution of the nerves of man with respect to number, size and myelination of their constituent fibers and the respective relations of such fiber constituents to the various receptive structures require more study. And why does stimulation of a nerve trunk always elicit pain or dysesthesia? This is familiar but a important fact.

Approaching somewhat nearer the subject of this discussion, the ganglia of the dorsal roots also present problems. Among these may be mentioned the important question of the possible ending in them of afferent sympathetic fibers with its obvious bearing on referred pains and possibly on the "antidromic" con-

duction of Bayliss, the meaning of the collateral branches given off within the ganglia by the processes of the ganglion cells and terminating in the ganglia, and last, but not least, the meaning of the various types of ganglion cells both as to form as shown by Dogiel and others and as to disposition of chromophilic substance. Considerable progress in this direction has already been made.

In regard to the roots, there are also moot points, some of fundamental importance. For example, there is the question of efferent impulses passing out along the dorsal roots and the allied question of the meaning of the dorsal root fibers originating from cell bodies lying within the cord. Are these fibers of universal occurrence or simply aberrant causal phenomena? It may be remarked, too, that such fibers as have been demonstrated by the method of Golgi do not arise from cells of the preganglionic sympathetic type but from cells of the ordinary somatic motor type located in the ventral gray horn. There is also the question of a low form of sensibility passing in by the ventral roots. Further studies of the composition of the roots in human beings, especially the dorsal, as to size of fibers and myelination are desirable.

At this point it might be well to allude to two subjects now beginning to receive more and more attention. One is the question of what may be termed sensory repercussion; i. e., sensations arising from peripheral changes caused not by ordinary peripheral stimuli but by efferent impulses from the central nervous system, coupled perhaps with the question of the possible antidromic activities of fibers belonging to ganglion cells whose central processes may or may not have been destroyed. Of the first, there are the examples, in exaggerated forms, of the proprioceptive thought theory of Watson and the interoceptive emotion theory of James-Lange.

The other subject, allied in some of its aspects to the last named, concerns the nature of the peculiar sensations, aroused under both normal and abnormal conditions, and known variously as dysesthesia, paresthesia and hyperpathia. As abnormal manifestations, they can apparently occur concomitantly with otherwise normal sensibility or with disturbance of almost any one of the various modes of sensibility. They may also arise, apparently, though perhaps with some variation as to character, in the presence of lesions not only in the peripheral but at any level in the central nervous system including the cortex cerebri. There are various theories regarding their causation. The first theory is that they are caused by irritation either of an afferent path, which irritation may be local, due to some lesion, or general, due to toxins, vasomotor disturbances, etc.; or irritation of an efferent path which may secondarily, by repercussion, say from a vasomotor disturbance, cause the sensation. According to the second theory the sensations may be caused by normal stimuli traversing an afferent path which has been damaged either by some local lesion or by some general abnormal influence (toxins, vasomotor disturbances, etc.). This possible explanation is not usually sufficiently taken into account. Stimuli are probably always passing along all afferent paths and would account for the persistence as well as variations of the abnormal sensations better than the first explanation. Stimulation by repercussion as in this first theory is also a possibility. The third theory considers the many forms of sensation as blends of various components, removal or weakening of a component perhaps bringing another into relief, so to speak. This is not an inhibition theory, as it simply presupposes a larger proportion of afferent impulses following a certain path than usual. The fourth theory deals with removal of inhibition, by injury or otherwise, of descending impulses; it is similar to Head's theory as to the cause of increased "affect" in certain thalamic lesions by removal of corticothalamic inhibitory impulses traversing corticothalamic fibers. This view throws a still greater burden on the obscure question of inhibition and assumes unproved descending paths to account for disturbances at lower levels.

It may not be inappropriate to remark that on the solution of these questions as to repercussion and as to the nature of the aforementioned sensations and pain, in all probability rests the physiologic explanation of the various sensations complained of by psychoneurotic patients as well as the still broader question of the mechanism of emotional disturbances, so important to the psychiatrist.

It may also be remarked that the conception formulated by Head as to the duality of sensory mechanisms has still, in spite of many criticisms and suggested changes of names, in many respects much to support it, in a broad way, both on the anatomic and the physiologic side.

When one considers the central nervous system, where the sorting out and rearrangement of the afferent impulses begins, one encounters as many difficulties. Do all the afferent root fibers split? The importance of this question has been shown by the discussion as to whether the character of the impulses carried by the descending splits of the afferent trigeminal root fibers are different from those carried by the ascending splits. This question has not been entirely settled either clinically or by the important observations by Windle that a certain proportion of the incoming fibers bend upward or downward without splitting. Do the descending arms of the dorsal spinal roots carry "sensation" or only afferent reflex impulses, and what is their length and location in various segments of the cord? In the ascending arms of the coarser-fibered medial part of the dorsal root, what is carried by those ascending splits which do not reach the bulb, and what relation may their varying lengths bear to the prevailing obscurity concerning tactile paths? Also what relation do they bear to the curious phenomenon that disturbances of dorsal column sensibility so often predominantly affect the apexes of the extremities, diminishing regularly as the trunk is approached? In this respect the distribution of cortical sensibility is much the same as that of its cortical efferent mate, the pyramid. Yet the sensations of movement of the larger joints must also reach the cortex. Why is vibratory (tuning fork) sensibility often less affected than position and movement? Such differences and the usually greater disturbance of the discriminative types of sensation in cortical lesions do not so much indicate, probably, a lack of projection of the less discriminative types on the cortex, but a different kind of projection, probably more diffuse—a characteristic that may obtain all along the line. The question of incomplete crossing must also be taken into account.

The substantia gelatinosa presents another obscure point, or rather, area. It has an intimate relation with the lateral, finer fibered, short-armed portion of the dorsal root which sends its (amyelinated?) collaterals or terminals into it, but which also sends some (myelinated) fibers through it and into the deeper dorsal gray. The gelatinosa also receives collaterals from the medial portion of the dorsal root. The axons of its cells appear to form short tracts lying laterally and perhaps dorsally to it (reflex and segmental integration?). If the all or none law did not apparently stand in the way, these arrangements might afford a convenient explanation for some of the phenomena of pain and other forms of affective sensibility, both their close connection with, and their separability in certain conditions from, other modes of sensation. In any interpretation of the gelatinosa it must be remembered that it as well as the dorsal column is one of the newer structures, increasing as the scale is ascended. Here also, as in higher levels involving synapses along afferent paths, one touches on problems of general neurophysiology which still await solutions.

Now actually touching on the subject of this discussion, unsettled questions relating to central tracts and paths arise. Any one who has looked over textbooks on the subject has probably been struck with the discrepancies between the various figures showing the location of the tracts of the cord, to say nothing of the higher levels. How often, for example, is the lateral pyramidal tract seen placed against the adjacent gray—and, by the way, how far down does the medial pyramidal tract usually run? I have had occasion to reprove severely an eminent and honored member of this Society for extending the dorsal gray horn to the periphery of the cord in his drawings on the board. The position of the vestibulospinal tract appears to vary in various books, etc. These variations, or inaccuracies, appear to arise from various causes, among which may be mentioned: (1) overschematization. (2) Carrying over in toto into human anatomy results obtained, experimentally or otherwise, on other forms. It is now becoming more and more clear that, valuable as are such results, the differences between the human and other nervous systems

must be taken into account; the value of comparative anatomy as an aid to understanding the human nervous system arises, in fact, from such differences. (3) Ignoring of the fact that the fibers of many tracts are more or less intermingled for part or all their courses. The lateral pyramidal and dorsal spinocerebellar tracts probably approach most nearly that ideal of purity so earnestly wished by the schematizer in anatomy as by the standardizer in social matters. (4) Ignoring of variations at different levels. Has any one seen illustrations showing the positions and sizes of each tract in each segment or even in a number of levels of the human spinal cord?

In connection with such level studies arises the possibility of separating the long from the short or spino-spinal tracts as a preliminary to a better understanding of the latter as to their possible rôles in (*a*) spinal reflexes, (*b*) as parts of polysynaptic long paths, possibly for diffuse conduction (protopathic, hyperpathic etc.?) or (*c*) as links between descending long neurones and efferent peripheral neurones. (5) Failure, in studying secondary degenerations, to take account of so-called "retrograde" degeneration.

In brief, there is, to my mind, a real necessity for the publication of careful studies of degenerations in the human spinal cord and brain stem in suitable cases by the Marchi or Weigert methods, the former preferably, and illustrated not by sketchy drawings, but by large reproductions of carefully executed photographs of many levels. Such studies would not only be immediately valuable, but also constitute records from which subsequent students could make further deductions and comparisons with other material. In such cases when possible, a careful and complete study should be made of all forms of sensation.

In Dr. Stookey's case one has a realization in man of the ideal experimental operation, for it was conducted with a maximum of skill and precision, and preoperative and postoperative sensory tests were made. I think the importance of thorough preoperative and postoperative sensory examinations of all modes of sensation, including viscera as far as possible, as well as of reflexes when possible, should be emphasized in cases like this which have such a high general scientific as well as practical therapeutic value. Then, if opportunity for postmortem study arises, the case can be exploited to its fullest value. Even without the latter, the case has the greatest value practically and scientifically in the light it throws on the pain and temperature paths.

Regarding the question as to whether the operative lesion corresponds with the area of secondary degeneration termed the lateral or dorsal spinothalamic tract, I suppose only autopsy could determine this exactly. It might be pointed out that if it does correspond completely, one might be driven to assume a short neuron or polysynaptic path for temperature. There is, more ventrally located, the tract known as the medial or ventral spinothalamic, but it is rather doubtful whether any of its fibers reach the thalamus in man. Some of its fibers may simply be approaching the lateral tract to join the latter higher up. Foerster figures the temperature path dorsal to the pain path in the cord. Rather general and perhaps vague a priori considerations would seem to favor this view.

Regarding the question of tract lamination raised by Dr. Stookey, indicated, as stated by him clinically but apparently not by the slides shown, it may be remarked that there does not seem to be any such indication on the slides shown except in the high cervical section of the cord with a lumbar crush and stained by Weigert's myelin stain. In this high cervical level the fibers in the spinothalamic area do appear to be more compacted. In the Marchi cord, high levels were not obtainable.[1] It may be remarked, however, that there may be a partial lamination, i.e., at any given level the fibers in that part of the tract farthest from the gray may be predominatingly from the more remote lower levels and those nearest the gray from the nearest levels. All these questions show the need of the more precise studies previously urged.

In conclusion, I should like to ask Dr. Stookey several questions, although some have been in part or entirely answered, not only concerning the present case, but concerning his other similar cases.

1. How closely can the area of the lesion be localized in transverse section of the cord?

2. Is the analgesia produced by the lesion complete, both as to topography and as to all kinds of pain, not only cutaneous pain but deep pain, i.e., pain produced by heavy pressure, by hyperextension and hyperreflection of joints and by striking bony surfaces?

3. How far is the lesion above the upper limit of the analgesia?

4. Is that upper limit sharply demarcated?

5. Is there any homolateral loss or change in pain near or remote from the lesion?

6. Are there any changes in pain (e.g., defense), vasomotor or pilomotor reflexes?

7. Are there any dysesthesias, paresthesias or hyperpathia?

8. What are the reactions to cold and hot? Is the application of either painful? Are the reflex reactions to them altered? If so, where?

9. Does the operative evidence point to the temperature path being dorsal or ventral to that for pain?

10. Is there any disturbance of any other form of sensibility on either the same or opposite side? Of touch? Of the dorsal column types of sensibility?

11. Are there any symptoms referable to the cutting of the ventral spinocerebellar tract, which, of course, must have been involved in the lesion?

Dr. Stookey (in closing): I hope Dr. Strong does not expect me to answer all his questions. I can answer a few of them. As I pointed out in the paper, analgesia is not complete for the total number of segments. If the analgesia begins in the eighth thoracic segment it can, for instance, skip the fourth lumbar and the others are complete. In one case, esthesias had developed about a month after the operation, the patient complaining of a burning sensation of the leg, in spite of the fact that she had a complete loss of pain. Temperature, when applied in extreme degrees, did not produce any differences in one segment. There was the same effect as occurred in the normal area.

As to whether or not there are any cerebellar disturbances due to the cutting of the cerebellar tract, I find no evidence of it. I have one or two clinical points that occurred in the series that I thought would throw some light on this particular thing.

As to the exact level of crossing, apparently this varies considerably. The place where you cut varies a good deal with the patient. Sometimes it is made at the fifth thoracic segment, and in some cases it begins at the seventh, and in others at the ninth, and in still others down lower, so that there apparently is variation in the cut as there naturally would be when you make a free hand cut in terms of millimeters.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Regular Meeting, May 14, 1929

Junius W. Stephenson, M.D., *in the Chair*

Unusual Sequelae in Chronic Encephalitis. Dr. Walter Bromberg (by invitation).

Dr. Bromberg presented a boy, aged 13, who was admitted to the Manhattan State Hospital in March, 1929, with the history of an acute illness characterized by fever, diplopia, mental dulness, drowsiness and choreic movements of the extremities, in 1922. The condition was diagnosed as epidemic encephalitis. He recovered

from the acute illness at the end of seven months. Since then there had developed successively parkinsonism, a Fröhlich syndrome, mild diabetes insipidus, behavior and emotional disturbances and vegetative changes. Several peculiar tics had also developed which, together with the conduct disorder, led to his commitment to the hospital.

Physically, he showed all characteristics of advanced parkinsonism (bradykinesia, hypomimia, retropulsion, etc.); oculogyric crises with the eyes looking upward and to the right; irregular, unequal pupils, sluggish to light, with no reaction in accommodation, and difficulty in downward gaze. He also had a peculiar gait; he walked with a skip on the right foot and at every second and third skip made a complete turn of his body to the left. His spontaneous gait, therefore, consisted in two skips forward, a complete turn of the body to the left, then two skips forward, a complete turn of the body to the left, and then two skips forward again. Another interesting feature was a "gagging tic" during which he inserted two or three fingers of the right hand into his throat until he gagged. There were several other tics centered around the oral zone. He had a marked tendency to chew on the threads that he bit out of blankets, clothes, etc. All movements were compulsive.

In addition to the emotional instability he showed peculiar speech phenomena; he frequently started a sentence almost automatically with a given phrase, which he kept on repeating; he said that he was compelled to repeat the phrase—a type of automatic reiteration.

The case was worthy of presentation because of the unusual movements. The tics centered about the oral zone were noted as being similar to those seen in regressed catatonic dementia praecox cases. The mechanism whereby these early behavior patterns come to the surface in encephalitis would seem to be analogous to that of conscious performance in catatonic patients. Apparently, in dementia praecox intrapsychic conflicts release the behavior patterns whereas in encephalitis cerebral lesions release these early reflexes.

DISCUSSION

DR. BROCK: This boy was studied at Bellevue. I do not know whether most neurologists would call this highly coordinated movement a tic. It is a compulsive, patterned, purposive act, which is much more complicated than the simple tic movement. On the other hand, it would seem that fundamentally it is a release of a motor "behavior" pattern. This conception becomes more interesting when one considers these episodes in connection with the intermittent dazed states—almost petit mal—which the boy exhibits. S. A. K. Wilson postulated removal of frontal lobe inhibition—a so-called "transcortical release"—for the major fit of epilepsy and for the petit mal attack. It is well known that in encephalitis the frontal lobe is almost invariably affected in these psychic cases. So it is not going too far afield to assume that here, too, one is dealing with intermittent removals of, or alterations in, transcortical inhibition, owing to involvement of the higher psychomotor level of Hughlings Jackson, i. e., in the frontal lobe. As a result, this peculiar motor pattern "escapes."

DR. STEPHENSON: It has only been since the advent of so much encephalitis that we have begun to regard these cases as organic. No doubt there is a good deal to be said on the purely functional side. For that reason I should like to hear from Dr. Lehrman.

DR. P. LEHRMAN: I will only point out that these organic cases have been studied, or an attempt has been made to study them, psychoanalytically by Dr Jelliffe in this country, and by others abroad. Psychoanalytic interpretation of the psychic effects of paresis and of encephaltis has also been attempted.

According to my feeling, one sees here a release, a paleopsychic release, just as one sees under the physical level a paleokinetic release.

DR. BROMBERG: In designating this movement a tic, I know that the term is not accurate, but I was led to it by the general definition of tic as a coordinated movement executed without the patient's voluntary control.

A Fulminating Case of Chorea. Dr. A. N. Bronfenbrenner (by invitation).

The number of cases of infectious chorea that have been carefully studied anatomically is still small, and the reported anatomic observations still seem to many unconvincing. Because of this, a careful study of another case of infectious chorea was undertaken. A thorough search for anatomic alterations throughout the brain stem and cerebral hemispheres was made by means of serial sections.

While no attempt is made to draw final conclusions as to the nature and location of the lesion in infectious chorea, it is, nevertheless, thought that the observations in this case are highly suggestive and seem to support the view that chorea is associated with a pathologic process in the basal ganglia, and more probably in the neostriatum.

S. A., a girl, aged 21, was admitted to the Mount Sinai Hospital on April 4, 1928, with a history of general malaise and a tingling sensation in the fingers of one week's duration, following which there appeared abruptly purposeless movements and twitchings in all extremities and in the facial muscles.

Examination revealed that the girl was well developed, with violent choreiform movements of the entire body. The temperature was 105 F. The spinal fluid was normal and under normal pressure.

The patient's restlessness continued, the choreiform movements became increasingly more violent and could not be controlled therapeutically. The temperature rose continuously, and when it reached 107 F. the movements had almost completely disappeared, but the patient appeared to be in a state of profound exhaustion and gradually sank into stupor. She died on the fourth day after admission.

Except for the somewhat larger volume, softer consistency and moderate obliteration of markings of the internal structures in the pons and cerebellum the brain and meninges appeared normal macroscopically.

Histologic study revealed the following: 1. Cerebral Cortex: Except for an occasional minute perivascular hemorrhage no changes were noted. 2. Corpus Striatum: The dominant anatomic alteration was a frequent and often pronounced perivascular extravasation, with mild adventitial infiltrations of small round cells. These infiltrations as well as the hemorrhages were more numerous in the neostriatum. Moderate gliosis was also found in the regions in which hemorrhage and infiltration were most pronounced. The ganglion cells took Nissl's stain poorly. 3. Thalamus: This structure showed changes similar to those in the corpus striatum, except that they were less marked. 4. Midbrain: The outstanding lesion was the perivascular hemorrhages. No frank inflammation or degenerative changes were noted. 5. Pons and Medulla: Particularly in the medulla, the most prominent histologic changes were massive perivascular infiltrations with mononuclear elements. The most advanced inflammatory process was found in the region of the nucleus ambiguus and in the tractus solitarius. (This may have accounted for the rapidly fatal termination.) 6. Cerebellum: This showed small glial rosets (circumscribed areas of gliosis) and numerous perivascular hemorrhages.

The heart showed a flabby myocardium and a number of small, rough, irregular, light brown masses, measuring about 5 cm., on the free edge of the mitral valve. Histologically, the lesion was considered nonrheumatic and as belonging to the indeterminate group.

Summary.—The report of a case of infectious chorea with an unusually stormy course and of brief duration with fatal termination was presented. Careful anatomic investigation disclosed undoubted evidence of an inflammatory process which justified the grouping of the case among the encephalitides. An unusual feature was the presence of inflammatory lesions in the hindbrain and the occurrence of only mild inflammatory changes in the basal ganglia. The latter, however, was the seat of numerous perivascular hemorrhages, which may be regarded as part of the inflammatory process, if one recalls the interpretation given by Globus and Strauss in their study of subacute epidemic encephalitis. These observers pointed out that in inflammatory conditions the exciting agent may produce either infiltrative or hemorrhagic reactions depending on the virulence and intensity of

the invading agent. Such variation not only may occur as a uniform reaction in the entire brain, but may be determined by sites of predilection in the brain stem. Thus, the medulla may react to the invading organism by infiltrations, while the corpus striatum and midbrain, which are sites of predilection for this disease, may react by hemorrhagic phenomena as the result of a more destructive process in the vessel wall.

Conclusion.—The observations in this case tend to support the view that chorea is most likely due to a disturbance in the normal anatomy and physiology of the neostriatum and allied structures.

METASTATIC CARCINOMA OF BRAIN AND CORD SIMULATING POLYNEURITIS. DR. HERMAN SELINSKY (by invitation).

M. G., a man, aged 43, was admitted to the Mount Sinai Hospital on Feb. 19, 1929. His past history was irrelevant. About seven months before admission, for a period of two weeks, he experienced pain in the substernal region radiating to the back. Three weeks later, severe constant pains appeared along the posterior aspect of both thighs, radiating to the hips and toes. This was followed by weakness in both lower extremities, so that for nine weeks preceding admission, he was confined to bed. About this time, he began to suffer from pain over both eyes and paresthesias at the top of the head. On several occasions he complained of diplopia and diminished hearing in the left ear. Somewhat later, the left side of the mouth began to droop; the tongue felt clumsy, and it was difficult for him to open the mouth. The eyes would remain half open during sleep. For the last five or six weeks before admission, bladder and rectal control were impaired.

The patient was first observed at a clinic in the West, where x-ray studies of the skull and spine, blood examinations and Wassermann tests of the blood and spinal fluid gave entirely negative results; diseased teeth were present, however. The diagnosis of polyneuritis with facial diplegia was made. Soon after returning home, he began to experience paresthesias, pain and advancing weakness of both upper extremities. Two days before admission he began to suffer from marked dysphagia. The intellect remained unimpaired.

On admission, he appeared acutely ill, and was incontinent of urine and feces. There was a decubitus ulcer on the left side of the sacrum, and signs suggestive of early consolidation at the base of the right lung. The speech was nasal. The right pupil was larger than the left and did not react to light, the left reacting only slightly; there was deviation, with some limitation of upward movement of the right eye. There were also: paresis of the left internal rectus; bilateral corneal hypesthesia; impaired hearing on the left, and bilateral peripheral facial paresis. The right palate did not move well. There was no definite weakness of the upper extremities, but some atrophy of the interossei. There was generalized atrophy and marked weakness of both lower extremities. The deep reflexes were diminished in the upper and absent in the lower extremities. The plantar reflexes could not be elicited. There was marked hypalgesia and thermal hypesthesia in both lower extremities involving the lumbosacral segments with almost complete loss of pain sensibility in the sacral segments. Vibration sense and position sense were also impaired in both lower extremities.

Lumbar puncture yielded a clear fluid under normal pressure, containing 31 cells per cubic millimeter of the large mononuclear type. The diagnosis of an extensive polyneuritic process was made, most likely inflammatory, but the possibility of multiple neoplasms was also considered. Total inability to swallow developed and it was necessary to feed the patient by gavage. The patient declined steadily, and a bilateral neuroparalytic keratitis developed. Pulmonary embarrassment followed and death occurred on the eighth day after admission.

Aside from marked dulling of the leptomeninges, the brain showed no gross abnormalities. Several roots of the cauda equina presented small globular swellings, which on section proved to be metastatic nodules.

Histologically, the brain, cord, cauda equina, cranial nerves and meninges showed carcinomatous metastases, chiefly along the meningeal spaces.

The primary tumor was situated in the right bronchus. Metastases were also found in lungs, spleen and right suprarenal.

Comment.—Dr. Globus and I called attention in a previous publication to the clinical picture, which is fairly typical of a generalized metastatic neoplastic process in the central nervous system. The presence of such a process may be strongly suspected in cases of acute onset of cerebral manifestations with the rapid development of neurologic signs of a disseminated character and symptoms of increased intracranial pressure without changes in the disks, with negative serologic observations and without febrile manifestations. Such a probability is strengthened by the progressive evolution of the neurologic signs and the appearance of wasting and asthenia out of proportion to that usually encountered in primary tumors of the brain. Under these circumstances careful search should be made for the primary focus.

DISCUSSION

DR. GLOBUS: The case presented by Dr. Bronfenbrenner is important from the anatomic point of view and because it throws some light on the localization of the lesion in chorea. In order to have a clear conception of the problem of localization, one must decide definitely as to the significance of the minute pathologic changes. From the lantern slides it was evident that the infiltrative inflammatory lesions were found mainly in the medulla and pons. There they were extensive. But in the corpus striatum, thalamus, midbrain and cerebellum the lesions were entirely different; they were mainly in the nature of petechial hemorrhages and only occasionally was an infiltrative process seen.

In a paper on subacute epidemic encephalitis published several years ago, Dr. Strauss and I suggested the possibility that in inflammatory lesions of the brain, if the invading organism is exceedingly virulent and intensive in its action, infiltrative lesions may not come into existence, and instead many hemorrhages will result from rupture of weakened blood vessels; these hemorrhages must not be regarded as purely mechanical; they are part of the inflammatory process. When lesions in the nature of infiltrations are found in other parts of the same brain, there is ample evidence that the hemorrhages are part of the inflammatory process. If one accepts these hemorrhages as part of the inflammatory process, one can interpret the significance of their location in the neostriatum, thalamus and midbrain as part of the disease process, and may say: Here is an inflammatory disease of the nervous system, most marked in the neostriatum, less in the thalamus, and still less in the paleostriatum, even though infiltrative lesions are not frequent there.

With this in mind it may, I think, be said that chorea is a disease most likely associated with alterations (inflammatory or degenerative) in the corpus striatum, and most markedly in the neostriatum. Some glial changes and a few hemorrhages were found in the cerebellum. These cannot be entirely dismissed; they may also play some rôle in the process.

The diagnosis of metastatic carcinomatosis of the brain was suspected and should have been made. If a roentgenologic examination of the chest had been made, the correct diagnosis would have been made earlier in the clinical course.

DR. NEUSTAEDTER: Will Dr. Bronfenbrenner state the observations of the pupillary light reflexes, spinal fluid and blood cultures?

If I am not mistaken, Thomalla reported microscopic observations in a case of chorea which are in agreement with the case reported. Thomalla also emphasized the fact that the most severe lesions are in the neostriatum, particularly in the small cells of the caudate and putamen. The hemorrhages in encephalitis are rather agonal and do not belong to the pathology of encephalitis.

DR. BRONFENBRENNER: I mentioned that the spinal fluid was normal. If I am not mistaken, blood cultures were made and gave negative results.

As Dr. Globus has mentioned, the question is whether there is a real difference between the chorea of encephalitis and Sydenham's chorea. There might be.

DR. GLOBUS: The pupillary reflexes were normal. The question whether this is a case of Sydenham's infectious chorea or of encephalitis cannot be answered

definitely. No one can draw a distinct line between infectious chorea and chorea on an encephalitic basis. Both are inflammatory and therefore encephalitic processes. The cause of one form of chorea is unknown, and one is only guessing about the cause of the other.

DISAPPEARANCE OF TREMOR IN A CASE OF PARALYSIS AGITANS FOLLOWING AN ATTACK OF HEMIPLEGIA WITH COMMENTS ON THE PRODUCTION OF THE TREMOR IN PARALYSIS AGITANS. DR. J. W. STEPHENSON.

A man, aged 60, had an advanced case of paralysis agitans and for three years had been unable to feed himself or turn over in bed. Five months before presentation, he was discovered at his usual waking time in profound coma, from which he could not be aroused. About six hours later, it was noticed that the tremor of the left upper extremity had disappeared, but in its place there developed violent spasmodic contractions of the left upper and lower extremities, sufficiently violent to displace the bed clothes. The patient gradually came out of the coma and the contractions disappeared after thirty-six hours, leaving the picture of a complete left hemiplegia with complete loss of motor power, hyperactive deep reflexes, ankle clonus, positive Babinski and Hoffmann signs, etc. Within ten days, improvement in motor power was noticed, and this continued until four months after the insult, when the patient was able to grip the hand firmly, put a cracker in his mouth, raise the arm above the head and push the bedclothes off and on. Close watch was kept for any sign of return of the tremor, but none was seen.

He then had another vascular accident which was not preceded by coma or spasmodic contractions. One month later, he was able to flex and extend the fingers slightly and to pronate and flex the forearm mildly, but there had been no return of the tremor. From the initial insult on there had persisted a marked mental change in the patient (aberration, fabrication and transitory delusional and hallucinatory states). He frequently had states of profound sleep bordering on coma. Yawning, though not so conspicuous at the time of presentation, for the first three months was a prominent symptom, and for several months it was necessary to keep a urinal constantly applied. Four months after the first insult, the Hoffmann sign had disappeared and the plantar response was equivocal. The persistence of the abnormal mental condition, periods of profound sleep, yawning and vesical disturbance was considered evidence of a destructive lesion of the prefrontal lobe where the site of the original vascular accident was thought to be.

With the restoration of pyramidal system function as manifested by return of motor power and disappearance of the Hoffmann and the equivocal Babinski signs, it was felt that the theory that the pyramidal system possesses an inherent tremor which is controlled by the striatal mechanism is disproved. It was contended that the theory that the tremor of paralysis agitans is produced by disease of the pallidum and that its disappearance in case of hemiplegia is due to the resulting spasticity preventing the tremor is also disproved, and that Wilson's suggestion regarding lower motor neuron diaschisis is not borne out in this observation.

Dr. Stephenson was of the opinion that neither paralysis agitans nor encephalitis was the proper condition under which to study tremors, the former because of involutional changes, the latter because of multiplicity of lesions. He thought that the nearest approach to the proper interpretation of tremors is a study of tremors as produced by proved intracranial neoplasms. He cited the rarity of rhythmic tremors in cerebellar tumors, and was of the opinion that the same is true of tumors near the red nucleus or rubrospinal tracts. It was contended that deep-seated tumors showing thalamic involvement must of necessity compress surrounding structures, but that clinical experience does not warrant the assertion that rhythmic tremors are concomitant with such localized neoplasms. Rhythmic tremors as a common observation in tumors of the frontal lobe was stressed.

When complete histories are available, it is believed that a proper study of them would show that the first clinical change in a patient with paralysis agitans would be in the psychic field, and this in collaboration with the rhythmic tremor to follow, and the study of the case reported caused Dr. Stephenson to suggest a

more thorough investigation of the anatomy and physiology of the frontal lobes. It was his conclusion that the tremor of paralysis agitans is caused by irritation, not destruction, of undiscovered centrifugal frontal pathways.

DISCUSSION

DR. JOSHUA ROSETT: In the study of the physiologic causes of abnormal movements, such as those of parkinsonism, chorea, the athetoses and dystonia, one must bear in mind the fact that they disappear in sleep and under anesthesia. The parkinsonian tremor also disappears, for a time at least, when the patient has suffered from hemiplegia.

From the disappearance of these abnormal movements in sleep, when sensory activity is inhibited, one may legitimately assume that they are a function of the sensory or the long reflex arc; this assumption is sustained by the fact that tremor ceases when the pyramidal tract is disabled. For if the afferent arm of the long reflex arc is constituted of the sensory nerve pathways which proceed to the cerebral cortex, the efferent arm of that arc consists of the efferent tracts of the cerebrum, of which the pyramidal tract plays most directly on the motor mechanisms of the brain stem and spinal cord.

There is, however, a discrepancy in the reports of different observers regarding the degree of permanency of disappearance of the parkinsonian tremor after the onset of hemiplegia. It seems to me that the reason for this discrepancy is a failure of the observers to take into account the following two factors: In a number of instances of hemiplegia, the pyramidal tract is not actually, certainly not entirely, severed by the hemorrhage, although for a time it may be entirely disabled. With the absorption of the hemorrhage and the consequent relief from the pressure exerted by it, the intact fibers of the pyramidal tract may recover their function. In such a case the tremor, which was absent for a time, will return. Another factor is the deceptive appearance of the patient in instances of satisfactory recovery from hemiplegia. I think that it has been fairly well established that the pyramidal tract is not the mechanism of posture. The latter is rather a function of certain nuclear masses in the brain stem and spinal cord. The cerebrocerebellar and striate mechanisms play on these nuclear masses and are conducive to gross changes in posture. Hence it is that in injury of the pyramidal tract some movement is preserved — preserved most in the relatively central joints, which under normal conditions are engaged in posture and changes of posture. Changes in posture of the relatively central joints brought about by the extrapyramidal systems may in hemiplegia easily simulate a high degree of recovery of pyramidal function. In such cases, however, a parkinsonian tremor will disappear permanently.

I cannot, therefore, agree with the statement frequently made that a parkinsonian tremor is a regression to a low form of movement. It is, perhaps, true that the ultimate nerve mechanism of tremor is to be found in the lower neuraxis, but so is all movement and all posture. It is, indeed, impossible to say which is a high and which a low grade of movement. All that one can say is that the tremor is, under the circumstances, a useless movement. In the absence — as a result of injury or inhibition — of the automatic and fundamental nerve mechanisms, the human sensory system becomes useless, and any motor expression of its activity then becomes abnormal.

DR. S. BROCK: The true physiopathologic explanation of tremor is surrounded with difficulties. I have had the advantage of discussing this case with Dr. Stephenson and of hearing Dr. Orton's paper last week. I think that Dr. Rosett is to be commended for a broad philosophic approach to the subject. But when he regards tremor and, by implication, other involuntary movements in terms of function of the sensory systems, and then infers that the pyramidal tract alone mediates these involuntary movements, I must register certain objections.

I cannot analyze the tremor of paralysis agitans or the movements of chorea and athetosis in such terms. One need not thereby deny that the sensory systems have a good deal to do with these involuntary movements. After all, one cannot

regard the nervous system in its entirety in seeking an explanation for tremor, but must limit one's inquiry mainly to the motor system.

There does not seem to be any question that certain cases of Parkinson's disease are beset by hemiplegia, and that the tremor disappears on the newly paralyzed side. After the expiration of a number of weeks, which are presumably the weeks of shock, the tremor reappears. Yet the patient has the Babinski toe sign, spasticity and other signs referable to a defective pyramidal tract. For Dr. Rosett to state that the pyramidal tract has really resumed function and that some other mechanism concerned with posture is responsible for the signs usually attributed to defective pyramidal tract function, is to assume a position beyond the facts. Again, if the pyramidal tract transmits the tremor impulses, why is it that the tremor is so often inhibited in a parkinsonian during the performance of a volitional act — a valuable point raised by E. D. Friedman. One can hardly conceive of one tract (namely, the pyramidal) mediating tremor-bearing impulses and voluntary motor stimuli, the latter inhibiting the former.

As I said in discussing Dr. Orton's paper, I still think that the matter can be approached in a simpler manner if one accepts Wilson's point of view. At least one can postulate a simple plan and then elaborate it with Dr. Stephenson's observations in mind, namely, that the tremor-bearing impulses are mediated by "released" anterior horn cells; that the diaschisis which throws out the anterior horn cells' function in hemiplegia interferes with the tremor-bearing impulses; that as the pyramidal tract disease regresses and the period of shock passes, the tremor reappears.

Such a concept regards tremor as a lower form of involuntary movement than the more complicated, patterned chorea and athetosis.

Even such a simple hypothesis as that of Wilson encounters difficulties. Why should cerebellar and mesencephalospinal and diencephalic mechanisms inhibit tremor? Why do such elaborate mechanisms exist to inhibit this curious rhythmic movement? Again, what is the "biologic need" for a tremor mediated by anterior horn cells? Are there undefined frontal tracts which also inhibit it? These questions I cannot answer. With Dr. Rosett, I think that one should keep an open mind on the whole question. Much more must be known before the phenomenon of tremor is clearly understood.

DR. STEPHENSON: If Dr. Rosett has any doubt about the ability of my patient to use his fingers, I should like very much, indeed, for him to be able to shake hands with him. I do not believe for a minute he would think it was a "postulatory squeeze."

AEROPHAGIA AS AN IMPORTANT FACTOR IN THE NEUROSES AND SUGGESTIONS FOR RELIEF. DR. GEORGE A. GOSSELIN (by invitation).

Aerophagia is a phenomenon which was commented on by Dejardin in the early part of the nineteenth century. Ewald, in 1876, comparing the physical and chemical characteristics of eructated gas, concluded that much atmospheric air must be swallowed by belchers. Experiments by Leven, Lesage and Barret with bismuth and x-ray demonstrated excessive swallowing of air in belchers. Mechanical pressure of gas in the stomach such as elevation of the diaphragm on the left, which is normally lower, causes a series of aggravating symptoms. The capacity of the thoracic cavity is lessened, with accompanying dyspnea, concomitant pressure symptoms and also interference with heart action. The frequent complaint of pain over or under the heart which beats against the diaphragm is explained in this way. Vaquez and Bordet definitely established that extrasystoles could be produced by artificially inflating the stomach. Torsion of the heart on its axis may also be a possibility in disturbed rhythm. The anxiety state accompanying such symptoms is thus well explained.

It has been commonly considered that aerophagia is a hysterical manifestation. G. Hayem, in 1898, 1902 and 1907, emphasized the fact that this symptom occurred more frequently among patients who were not obviously hysterical. The large percentage of office patients who suffer from psychoneuroses seems to make this

study worth while because of the evident involvement of the sympathetic nervous system, with its widespread reactions.

In the treatment it is important to eliminate salivation, which Hurst believes is a frequent source of aerophagia. Salivation is caused by local irritation in the mouth and esophagus, and also, probably, reflexly from irritation of the abdominal sympathetic.

Results of the exhaustive researches of Cannon on the flow of the digestive juices are well known. Irritation of the abdominal sympathetic from visceroptosis must also be considered. Langdon Brown, in his Oxford lectures, upholds this measure, and quotes Hurst, Lane and others to support his contention. Tyrell Grey and Pirrie, quoted by Brown, state that stasis from sympathetic irritation tends to increase visceroptosis, and this in turn causes more drag on the mesentery. Abdominal support, while it may not raise the viscera, improves tone by increasing intra-abdominal pressure. The reflex vasodilatation which, I believe, occurs through vasomotor irritation involving the splanchnic circulation, thereby lessening intracranial blood supply, may explain the pressure sensations, dizziness and flushing as well as numbness in the extremities. Purely reflex phenomena such as globus hystericus can be stopped almost invariably by abdominal support. In this connection one might mention the prevalence of Chvostek's and Hoffmann's signs together with general increase in the reflexes. This brings to mind the possibilities of biochemical and metabolic disturbances referred to by Dr. Timme in his studies on the glandular syndromes.

I have adopted the medical suggestions of Leven, which consist of the administration of small doses of sodium bromide taken with food twice daily, because of its sedative effect on the abdominal sympathetic, and the administration of bismuth subcarbonate from three to five times a day, which Leven found allays salivation and also seems to have a sedative effect on the sympathetic. These measures may seem too simple and commonplace, but their use, with the other therapeutic procedures mentioned, has definitely established their remarkable value. As a psychotherapeutic suggestion, a simple and clear explanation of this mechanism given to a psychoneurotic patient serves to dissipate some of his fears and uncertainties by giving him a concrete cause for his numerous symptoms.

DISCUSSION

DR. STEPHENSON: I was interested to hear Dr. Gosselin speak of the simple measures in the treatment for aerophagia. I have had occasion to try his bromide treatment in these cases, in most of them with undoubted success.

DR. IRVING PARDEE: As I was reading Dr. Gosselin's paper, it occurred to me that it might be approached from two points of view. The first was the mechanical effect of the distention of the stomach with gas. This causes a certain amount of pressure on the abdominal sympathetic system, and also pressure on the suprarenal glands. The second was certain psychic considerations. For example, children often belch for fun. Older children belch because of the pleasant noise. It may perhaps even give them a slight sense of power, in some way to enhance a flagging ego lost on the altar of a maladjusted personality. There is no doubt that a delightful sense of power comes from a satisfactory, adequate belching. To some belching is habitual. They have no desire to stop it. And there may seem to be no neurosis back of it. It may be just a habit, formed in early childhood, which has persisted. It would be interesting to know the psychoanalytic interpretation of this problem. No doubt each case has its own mosaic.

A question which comes to my mind is perhaps the relationship between the passing of flatus and aerophagia. At times it seems as though there was some relationship between those two functions. Here, again, one may be dealing with a purely neurotic manifestation or a question of muscle tone.

Imbued as we are with the analytic interpretation of so-called neurotic symptoms and because of the immense knowledge which we have obtained of late on that subject we hesitate at times to approach even the organic or possible organic causes for some of these symptoms.

There is no question that there is an integral relationship between the vegetative nervous system and the stomach and intestines. Their functions cannot be carried on without adequate control from the vegetative nervous system. Again, one knows of the close relationship between the vegetative centers in the brain (thalamus, subthalamic region) with the vegetative centers in the intestines.

Then one knows that there is a close relationship between body chemistry and the vegetative nervous system. Metabolism is no doubt controlled, as far as its neural mechanisms are concerned, through the vegetative nervous system. And the body metabolism is a function controlled by the glands of internal secretion. These, I appreciate, are somewhat vague generalizations, but they are of considerable importance, because it is the line along which one will have to think in order to come to a final decision.

Recently there have been some interesting reports in the literature, starting with the brilliant discovery by Collip of the hormone of the parathyroid gland. This hormone controls the chemistry of calcium in the blood. Patients who have disturbed calcium metabolism can be treated adequately and hopefully with parathyroid extract-Collip. These patients with parathyroid disturbance have as an outstanding symptom a muscle hypotonia. That hypotonia is not only in the muscles of the arms and legs and body, but it may be also in the intestines. This muscle hypotonia, according to the case reports, results in a great intestinal disturbance and disturbance of the digestion of fats. Recovery in these cases is obtained by large doses of calcium combined with parathyroid extract-Collip.

What other chemicals of the body may be disturbed, producing digestive and other symptoms, at the moment, considered psychogenic, one does not know. The possibilities are limitless and await the investigator.

In treatment, the points elaborated by Dr. Gosselin are of great interest, and will bring an additional therapeutic approach. Owing to the extreme toxic disturbances often observed during the prolonged administration of bromide, in fact even with the short administration of bromide, I have developed somewhat of an obsession against the use of bromide, and rarely use it except in emergency. I find that phenobarbital does practically what bromide does without any danger from toxic symptoms. The rash which results from phenobarbital is very mild and clears up readily; toxic symptoms involving the mind are rare, but they are common after the administration of bromide.

Additional therapy in these cases which Dr. Gosselin has studied so carefully does not appear indicated, except that many of these patients who present a positive Chovstek sign with muscle irritability and myedema are on the borderline of spasmophilic cases in which the biochemical background is a disordered calcium metabolism. These patients would be aided also by the administration of large doses of calcium and parathyroid.

DR. THEOPHILUS P. ALLEN: I have two cases in mind that seemed to me to illustrate one of the psychic mechanisms underlying certain types of aerophagia. The two persons treated were noisy aerophagic patients; both were past middle age, and both had recently acquired aerophagia. One was suffering from cardiac decompensation, the other from angina pectoris. The chief complaint was "gas," and they were unaware of the cardiac disease. I carefully ignored the "gas" and informed them that they had heart trouble and must stay in bed. Both immediately ceased belching and did not resume it while under observation, for six months in one case, and for three months in the other; the latter died. That was rather striking and seemed to me to indicate that in these cases the noisy belching was simply a method of notifying the community that they were suffering, a modification of the mental mechanism that makes one groan.

In line with what Dr. Pardee says about the relationship between flatus and aerophagia, I have in mind a case of a consciously masochistic male who swallows air, fills his stomach, gets a pain from it and then proceeds to relieve himself by passing gas per rectum.

DR. LE WALD: From a roentgenologic standpoint I am satisfied that there are two types of this condition, one purely esophageal. I have seen cases in which

tremendous noises were evoked with no air actually in the stomach. I know Dr. Gosselin thought that was one of the conditions that had been observed, and I can confirm that. Other patients actually swallow the air and then eruct it from the stomach with a sudden opening of the cardia, and an explosive noise. There is no question but that the air is swallowed. We have all examined patients and in a few minutes after a meal noticed this excessive amount of air in the stomach, so that it is not a result of fermentation.

I am sure I have seen it in both the enteroptotic and the opposite type, the sthenic type in which it is associated with lesions of the gallbladder, just as much as at times with the enteroptotic person with gastric retention. And I am sure that a belt has relieved many of these patients. I am sure that I have confirmed that observation.

DR. GOSSELIN: As regards yawning and the resulting mechanical pressure on the suprarenals, I have learned something from Dr. Pardee's comments. This suggests another biochemical factor in disturbances of the abdominal sympathetic.

The question of flatus is a disputed one. Alvarez does not believe that a great deal of swallowed air reaches the intestines. Leven, however, lays particular stress on this phase of aerophagia, and comments at length in his work on "les deux douleurs coliques" due to distention at the hepatic and splenic flexures. There are also several other intestinal symptoms that I have not had time to cover, such as hemorrhoids from exhaustion or inhibition of the abdominal vasomotor system.

I agree with Dr. Pardee on the toxic effect of bromides, but I use sodium bromide in small doses, as low as 5 grain (0.324 Gm.) twice a day, for a limited period. In this way one is frequently able to get the patient out of a rut, and to break the vicious circle. Physical culture, abdominal support, etc., should also be carried out to benefit the patient further by improvement of intra-abdominal tone.

Personally, I disagree with Dr. Pardee on the action of phenobarbital in place of bromide in aerophagia. I have tried it without as good results. Dr. Pardee's comments on spasmophilia suggest the possibility of its origin in the aerophagia of nursing infants. In Leven's work considerable attention is paid to belching in infants. This condition may possibly initiate irritation of the abdominal sympathetic which plays a rôle in spasmophilia of later life.

I am pleased to have Dr. Le Wald confirm my remarks as a result of his x-ray experience.

CHICAGO NEUROLOGICAL SOCIETY

Annual Meeting, May 16, 1929

LOYAL DAVIS, M.D., *President, in the Chair*

PARALYSIS FOLLOWING ANTIRABIC INOCULATION AND OTHER FORMS OF VACCINE AND SERUM THERAPY. DR. PETER BASSOE.

Dr. Bassoe first related the clinical history of a case of paralysis occurring during antirabic inoculation, the histologic features of which were presented by Dr. Grinker. The full report of this case will appear in the ARCHIVES. After reviewing briefly the literature of antirabic inoculation paralysis, Dr. Bassoe called attention to the curious fact that during the twelve years which have elapsed since epidemic encephalitis became prevalent numerous reports have been made of encephalitis following vaccinations against smallpox and after measles and chickenpox, and of various central and peripheral paralytic reactions from prophylactic and curative serum and vaccine therapy. The latter reactions are usually a manifestation of so-called serum sickness which has been known for thirty-five years. Urticaria, angioneurotic edema, back and joint pains, fever and leukopenia are the main symptoms. It is reasonable to suppose that the paralyses which sometimes occur, particularly after the use of antitetanic serum in adults, are due

to edema in or around nerve structures. There have been also cases following inoculation against typhoid, scarlet fever and diphtheria. A brief review of the literature was given.

HISTOPATHOLOGY OF RABIES AND OF A CASE OF PARAPLEGIA AFTER ANTIRABIC INOCULATION COMPARED WITH THAT OF CERTAIN FORMS OF ENCEPHALITIS. DR. ROY R. GRINKER and DR. PETER BASSOE.

This article is to be published in full in the ARCHIVES.

DISCUSSION

DR. DOROTHY RUSSELL, London Hospital: I should have liked to have heard Dr. Grinker give Trumbull credit for having been the first to describe this condition. He described it before Dr. McIntosh, but the material was not published at this time because of the scare it might give, and the boost it might give to the antivaccination propaganda. Actually, although he did not get into print first, his communication was available to the government before any other.

I do not think that Dr. Grinker is altogether in accord with Dr. McIntosh in regard to the transmission of the disease to rabbits. However, there is a great deal of controversy about the matter in London, and the Staff of the Pasteur Institute is much against the idea that this form of encephalitis has anything to do with vaccination at all. They think that the vaccination acts by stirring up a virus that is already present in the patient, apparently in the upper respiratory tract.

DR. FRIEDRICH HILLER: In Wiesbaden, Spielmeyer, of Munich, read a paper on postvaccinal encephalitis. He stated that postvaccinal encephalitis is different from any other kind of encephalitis; he claims especially that there is no typical softening. There is no definite proof yet that vaccine itself is the cause of that encephalitis. Another point which Dr. Grinker mentioned is that the infiltration in rabies vaccination occurred around the arteries. If this is true and one certainly must believe it from the pictures, I think it also must be proved by further investigation. It would be rather surprising, for one is accustomed to see infiltrations and softenings principally around the veins and not the arteries.

DR. H. DOUGLAS SINGER: It is noteworthy that a number of different types of inoculation were used. Some, like rabies, were presumably of an attenuated virus; others were of horse serum, antidiphtheritic, etc. Is it intended to suggest that the pathology is the same for all of these? From the manner of presentation one gathers the impression that it is suggested that the picture is alike in all. A priori it would seem that there should be considerable differences.

DR. SIGMUND KRUMHOLZ: I recall a case, seen several years ago, that was very similar to that reported by Dr. Bassoe. A young man was bitten by a rat one night, and thought that possibly he would develop rabies. He was taken to the Pasteur Institute where antirabic serum was injected. After the ninth injection he became paralyzed; on physical examination he presented a picture of transverse myelitis: flaccid paralysis of the lower extremities, incontinence of urine and an anesthetic level. I do not now recall how long the paralysis persisted, but in the course of a few weeks he recovered completely. The patient is neurotic and from time to time appears at my office with symptoms characteristic of a vasomotor neurosis. This rather substantiates the conception that serum therapy will produce angioneurotic edema in this type of person.

DR. VICTOR E. GONDA: Can this condition be produced by animals?

DR. PETER BASSOE: The case described by Dr. Krumholz is like the one I have reported and the person he described is just of the type who would have this syndrome.

I had an interesting conversation with the elder Dr. Lagorio and asked him in regard to the incubation period and the symptoms. He has had a very long experience, and he states that he has never encountered any paralysis. He is

convinced that the reason such things occur is because the treatment has become commercialized, and too machine-like; it must be individualized. When the work is done in a wholesale manner, persons with small bites are treated in the same manner as those with large bites. If there are bites on the face and lacerations, such as occur from wolf bites, a higher concentration should be used; if this is done in cases of small bites the patients are more liable to have trouble. Lagorio prefers the carbolized vaccine. He is convinced that he has seen one case of genuine rabies with an incubation period of one year. It occurred in a child from Morocco, Ind., who had a clear history of having been bitten by a mad dog a year before.

DR. ROY R. GRINKER: I am much indebted to Dr. Russell for setting me straight regarding the first observer of this type of encephalitis.

In regard to the question of infiltration only along the arteries, the section I showed happened to be of an infiltration about an arteriole. What I implied was that the infiltration occurs around the larger vessels, around the large veins and arterioles, and not around the capillaries.

Dr. Singer asked whether the various vaccination disturbances are alike. I can only say that those that have been seen following cowpox vaccination and those following rabies vaccination have been clinically very similar and pathologically almost identical. That does not mean that the virus is identical. The reaction in rabies is pathologically very similar to that in epidemic encephalitis, but it may be due merely to the inability of the nervous system to react other than in one manner to a virus infection. The same thing occurs in an acute multiple sclerosis, which often is difficult to distinguish from epidemic encephalitis; probably it is a different virus in each case. More work such as McIntosh and Levaditi are doing is necessary. I know of no cases that have been studied following scarlet fever and varicella. Encephalitis following measles has been studied, with similar observations. The serum reactions are probably not virus infections.

PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, May 10, 1929

EVERETT S. BARR, M.D., *President, in the Chair*

THE PSYCHOLOGIC ORIENTATION IN INDIVIDUAL ADJUSTMENT. DR. MORRIS S. VITELES.

The outstanding difference between psychology and psychiatry is in the methods employed in individual analysis. A survey of the work of the psychologic clinic in the treatment of children who are maladjusted in school, in the home, etc., and a survey of the work of the industrial psychologist in promoting the adjustment of the worker show that the chief characteristic of the psychologic approach is its objectivity. The psychologic test for measuring individual differences, on which he depends so largely in his clinical work, reflects the objective point of view of the psychologist. Dissatisfied with the unstandardized objective judgments on human personality used by physicians in the diagnosis of mental status, he has developed standard, graded instruments for the measurement of ability and temperament capable of administration under controlled conditions and of objective scoring. It is in this respect that psychology differs most from psychiatry. The psychiatrist still leans heavily on uncontrolled conditions; on observations not subjected to careful measurement, and on interpretations of mass data that may be too largely influenced by a point of view reflecting more the bias of the individual worker than the expression of a general principle subjected to verification through the controlled observation of random, unselected samples of the behavior which is being investigated.

It is true that there has recently been a belated and almost grudging recognition of the value of these psychologic instruments on the part of the psychiatrist,

but it is a half-hearted acceptance that implies no true adherence to the ideal of complete objectivity in measurement toward which the competent psychologist is striving—as is the scientist in every other field. There is a difference in emphasis, on the part of the psychologist, on the perfection of the tool as an objective measurement instrument; on the part of the psychiatrist, merely its acceptance as an aid in subjective diagnosis. Even today there is little appreciation by psychiatrists of the exact controls which govern the standardization and validation of the psychologic test.

The inherent objectivity of the psychologic approach in the measurement of character, as distinguished from the inherently subjective approach of the psychiatrist, is illustrated in the attitude of the two groups toward the analysis of the so-called temperamental traits. It is true that there is as yet no altogether satisfactory method by which "the psychologist or anyone else has been able to measure these traits." Because they cannot now be measured is no reason for assuming, as is so consistently assumed in psychiatric literature, that they never can be measured. Insofar as these qualities may be even more essential for adjustment in many cases than are the qualities of competency, to the same extent it becomes necessary that objective methods for their measurement be devised. The psychologist's continued absorption in the preparation of objective tests for the measurement of these traits is another reflection of the faith which he places in objective instruments and his hope for a continually decreasing dependence on subjective judgment, such as at present must be used in measuring the temperamental traits.

This insistence on promoting objective tests for the measurement of character traits, whether they be mental abilities and defects or traits of temperament, does not imply that the psychologist can, as yet, depend exclusively on psychologic tests for data concerning the mental make-up of a person.

I have no hesitation in agreeing with Anderson's criticism of the psychologist who considers the psychologic tests as the sole or final selection agent and with his statement that "the interview if properly conducted and intelligently interpreted opens up knowledge concerning the individual's past history and ways of behaving that furnish the most fruitful basis for judging what his future adjustments are likely to be."

However, in the method of answering the question of what is a satisfactory interview and when an interview can be considered to have been properly conducted and intelligently interpreted, the objective slant of the psychologist is again revealed. He measures the effectiveness of the ordinary interview conducted by those responsible for selection in industry and finds, for example, that the agreement among twelve sales managers on the fitness of fifty-seven candidates were placed in a hat, shuffled and drawn by a blindfolded man.

Other examples could be cited to demonstrate the objective character of psychologic approach in distinction from the individualistic and subjective character of the methods of study used by the psychiatrists. The technical developments of a science and its possible contributions are in a large part influenced by what may be called its philosophic foundations. Similar as are the aims of psychology and psychiatry, their differences in philosophic outlook are bound to reflect themselves in the character and effectiveness of the contribution of each. The differences in philosophic foundations are many. The most important of these is the psychologic orientation toward the normal as contrasted with the psychiatrist's orientation toward the abnormal.

Much as he has tried, the psychiatrist seems never quite to get away from his experience with groups of seriously disordered persons whom, in the early history of psychiatry, he was called on to treat and who, even today, in the case of many psychiatrists constitute the bulk of his practice. It is true that a number of psychiatrists appreciate this situation and have taken steps to remedy the difficulty. Recognition of the problem is implied in the description of the mental hygiene movement by its leaders as concerned "with not only the abnormal but the normal." In actual practice, however, there still persists an abnormal orientation.

The prevalent attitude of psychiatry and of the psychiatrist is perhaps better stated in the words of Stevens, who wrote: "If something is wrong with the mind

in industry, to whom should industry turn for help if not to the psychiatrist with his trouble finding outfit of 'complexes,' 'obsessions' and 'delusions.'"

Stevens has here chosen a happy phrase. It is in trouble findings, whether it be in industry or elsewhere, that the psychiatrist seems most competent. It may be said that he tends to look at human conduct through misshapen lenses which tend to transfer the picture—to bring out defects at the expense of beauties, one might almost say the perfections of human character. In the consideration of the individual, if the lenses show nothing more serious, they revealed a perverted tendency on the part of the individual to fall in love with his mother or to subordinate himself to his brother. Everything else that may be seen in the picture of the mental life of the individual seems blotted out by the psychiatrist's absorption in the abnormalities of conduct which he has observed in the psychopaths to whom his interest has until recently been largely confined.

The psychologist, on the other hand, brings toward problems of adjustment a common-sense orientation toward the normal. He comes as an observer trained in the interpretation of certain facts which are made basic in any judgment which he may express. He comes prepared not to see primarily the abnormalities of human conduct, the freudian obsessions, irrational reveries, etc., but to study under controlled conditions normal human behavior and such deviation as may exist in well authenticated sampling, and to arrive from the study of these at principles concerning the causes of human conduct capable of application to promoting the welfare of the individual.

DISCUSSION

DR. SAMUEL W. FERNBERGER: It seems to me that there is apt to be a certain amount of criticism of this paper by psychiatrists. There might also be a certain amount by some psychologists, because there are psychologists of repute with a clinical reputation who seem to be willing to make a diagnosis on the basis of a single test score. I might even mention a psychologist of repute who some time ago published an alleged series of vocational guidance tests for work for high school graduates to determine whether they should go into engineering or not. These were to be sent to the subjects, answered in writing and sent back to the examiner who would make a diagnosis without ever having seen the subject.

On the basis of a certain amount of pressure from the American Psychological Association these tests were withdrawn from the market. The situation I have described is no worse than diagnosing by group testing as employed by some psychologists who seat 100 persons at tables and give them the tests as given at the University of Pennsylvania now without having seen one of the candidates personally. In other words, one must be cautious as to the kind and mode of application of psychologic tests. I agree with Dr. Viteles in insisting on an individual examination for diagnosis, an individual examination in which one considers the final score and the qualitative picture. It is necessary to know how the subject goes about his procedures.

Regarding the use of instruments, certain things have been designed for particular purposes. Psychologic tests were used in the army. There were not enough psychologists or psychiatrists and not enough men with psychiatric or psychologic training to examine the candidates. Some sort of preliminary sorting had to be made. To sort out the lower group (and the size of that group was to be determined by consideration of how many psychologists and psychiatrists could make examinations), and for that purpose the tests were excellent. To apply these group tests now and make them a basis for a diagnosis of mentality is to use them for a purpose for which they were never intended; failure is bound to result.

DR. ALFRED GORDON: I feel that Dr. Viteles' paper is a challenge to psychiatry. If the psychologic clinic deals with the behavior of a boy, for instance, who committed an act or who has criminal tendencies—is not the only approach in settling a question of this kind through pathologic means? Is it not true that in studying anatomy only by tracing pathologic conditions can one formulate an opinion on normal conditions, as for example degenerate tracts of nerve fibers. The behavior of a boy, his manner in responding to questions, his manner of response

to stimuli, the emotional expression of his face—these are all objective signs and not subjective as the speaker would have us believe. Personally, I do not think that measurements with instruments such as the galvanometer will determine behavior. I believe that through morbid psychology, normal psychology can be learned in an efficient manner.

DR. C. B. FARR: As to the objective study of emotional reactions, psychiatrists have, I think, an appreciation of the value of objective methods (e. g., the psychogalvanometer). Dr. Gordon has brought out the point that psychiatric cases may be regarded in a sense as experiments that give one an insight into the normal.

DR. MORRIS S. VITELES: In insisting on his ability to diagnose feebleminded-ness at sight, Dr. Burr emphasizes one of the weaknesses of psychiatry, its dependence on a subjective unverified observation. The purpose in developing psychologic tests is that of getting away from just such subjective diagnosis. In the case of feeblemindedness, the value of diagnosis at sight is well illustrated in a recent experiment in which psychologists and psychiatrists were asked to judge the mentality of fifty children whose pictures were shown to them. Twenty-five of the children were definitely normal and twenty-five definitely feebleminded. The judgments made by the psychologists on the basis of an examination of the photographs were as valid as if the photographs had been turned upside down and the backs instead of the faces of the photographs had been observed.

PSYCHOLOGY IN ITS RELATION TO MENTAL HEALTH CLINICS. DR. WILLIAM C. SANDY.

Psychology, at least so far as its relationship to psychiatry is concerned, has long constituted a controversial issue and continues from time to time to give rise to heated discussion in certain circles. Realizing this fact, it was with considerable hesitancy that I consented to discuss even the more limited field of psychology in mental health clinics. Furthermore, for the same reason, it has seemed wise to dwell somewhat at length on some general aspects of psychiatry and psychology in order that a suitable background may be provided for the consideration of the usefulness of psychology in mental clinics.

Psychometric examination has been one of the phases of psychology that have given rise to considerable difference of opinion. While it is rather generally accepted that mental testing by an experienced psychologist has a definite rôle as a laboratory procedure, extravagant claims by immature testers and diagnoses ventured by those who know no other criteria have caused many to question the value of the intelligence quotient.

Perhaps because it is a rather young professional subject, the limitations of psychology have not yet been very well established, and this has also resulted in considerable acrimony. Psychiatrists have observed certain psychologists tending to enter fields long regarded as purely medical, such as diagnostic determinations and therapeutic applications. This is not desirable, for psychologists not only do not have the requisite broad general training in basic principles required of physicians, but they have not developed that confidential relationship that exists between patient and consultant, protected by the well established system of medical ethics. On the other hand, some psychologists have professed to believe that psychiatry does not constitute a medical speciality, that psychiatrists are poorly trained, presumptuous in their claims and make no real contribution. Possibly this is merely a smoke screen or defense reaction. At any rate, one of the popular psychologic syndicate writers daily contributing to various papers articles on "keeping mentally fit" never mentions psychiatry, but occasionally by inference credits the physician with some usefulness.

Psychology, however, has gradually been coming to its own. While, as a name, "psychology" is a popular expression more or less misunderstood, it has been more and more clearly defined by higher educational requirements and by the increasing demands for service. The World War established the value of psychology in the selective service operations. Since then, psychology has been called on by the schools in the organization of special classes; in clinics such as those for

child guidance, the general mental clinic and the classification clinic of Pearce Bailey; in industry, in the employment of workers, in reassignment to more suitable positions or salvaging workers in the determination of the causes of labor difficulties and so on. Of late, there has developed renewed interest in psychology in the army, and formal tests are, in certain branches, being utilized in the selection of recruits and in ascertaining those who may be qualified for special service. The public press has made frequent references within the last few months to the endownment of $7,500,000 for an institute of human relations at Yale University. As projected, psychologists will combine with sociologists, biologists, economists and their colleagues in law, medicine and psychiatry in a study of man himself.

In at least one state the qualifications of psychologists who may act as examiners in the commitment of mental defectives are legally defined and psychologists and psychiatrists frequently certify in the same cases.

In the realm of higher research, psychology has contributed in the past and continues to contribute much of value in the study of mental mechanism and behavior.

In fact, there will probably be quite general agreement to the proposition that psychology can and does make valuable contributions in several respects. It has evolved fairly stable and broadly applicable methods of determining intellectual capacity, thus definitely entering into the diagnosis of mental defect. In a similar way, it is prepared to advise as to special bent or ability. Psychology has shown itself particularly successful in determining and analyzing special defects, and in advising the necessary educational modification, such as in those peculiarly interesting cases manifesting the inability to read. Psychology has even assisted in explaining the content of the psychoses and psychoneuroses by methods akin to the psychoanalytic school.

Psychiatry and psychology are really team mates with in many ways similar objectives but different though related functions. For instance, psychiatrists, unless especially trained and experienced in psychologic fields, should hesitate to pass on purely psychologic questions such as the determination of intellectual capacity. In the commitment of mentally deficient persons, if possible physical and mental disease has been accounted for, psychologists might be more competent to sign certificates than some psychiatrists who, as pointed out by Healey in 1922, in many quarters are not even familiar with the critical methods of determining special mental defects or the best terminology in use. Certainly the diagnosis of mental defect is poorly based unless supported by a competent psychologic opinion.

Psychologists, on the other hand, should avoid recommending procedures in cases without physical and mental consideration by physicians who are preferably psychiatrists. Disregard of the need for psychiatric or at least medical advice seems likely to be the tendency of the so-called "clinical" psychologists.

It is possible that in certain phases of psychoanalysis, psychologists might be as effective as psychiatrists practicing this particular method. But therapeutic activties are certainly outside the field of the psychologist. Some self-styled "clinical" psychologists, practicing as psychoanalysts, have undoubtedly in many instances accomplished more harm than good, as has very likely been the case with psychiatrists limiting their practice to the same field, with possible neglect of thorough physical and mental examination.

Adequate training in both fields (psychiatry and psychology) is impracticable for most of us, hence the importance of teamwork. It is believed, however, that with the growing recognition of the need of the study of the patient as a whole, rather than solely from a disease entity standpoint, more attention should be paid psychology in the medical school curriculum. This is especially true in any postgraduate psychiatric courses. If the time ever comes when psychiatrists will qualify as specialists through standardized postgraduate work, it is probable that psychology will enter largely as a major subject.

Psychologists therefore have well established spheres of usefulness, at least in research, education and as assistants or technicians in such clinical-diagnostic activities as mental clinics.

So far as the mental clinic activities in Pennsylvania are concerned (at least those under the auspices of the Bureau of Mental Health), the services of psychologists are utilized in a variable degree, partly due to lack of personnel and sometimes apparently because of failure to appreciate the need.

The clinics themselves vary in organization. There are a number manned wholly by state hospital personnel. These are apt not to have the benefit of the services of a qualified psychologist. A second type of clinic has its consultants partly from the institutions and partly from the bureau. This type of clinic is apt to be supplied with psychologic service from the bureau. A third type of clinic is supplied with both a psychiatrist and a psychologist from the bureau.

There are four psychologists on the staff of the Bureau of Mental Health. They are field representatives of the section on clinics and extra-institutional supervision. They are not only qualified as psychologists, but are experienced in family case work, field investigation, history taking, parole supervision, special class activities and allied topics. They are valuable, therefore, in many ways other than as psychologists. Through their initiative and organization ability, the interests of the mental clinics and their expansions are constantly being promoted. They function in the clinics, in preclinic and postclinic investigations, in initiating community supervision of patients, in investigation of the waiting lists to institutions for mental defectives and in special examinations of such as delinquents in institutions.

From the clinic reports filed in the office of the Bureau of Mental Health, it is evident that those clinics are the most effective agencies in which there are both psychiatrists and psychologists; in which those workers have a mutual understanding of their respective fields, and in which there is cooperation in conclusions reached and recommendations made. For instance, seldom if ever is the diagnosis of mental deficiency made without a thorough psychometric survey. It is equally true that the physical and psychiatric aspects are not neglected. Furthermore, in cases of mental deficiency in particular, the psychiatrist utilizes the training and field experience of the psychologist-social worker in rendering a well balanced plan for the treatment of the problem presented by the patient.

DISCUSSION

DR. F. H. ALLEN: The contributions of the psychiatrists and psychologists can be best made in the field of understanding behavior when there is a willingness and ability to work together so that each may benefit by the point of view and experience of the other. There is great difficulty in relying solely on measurements gained through the use of psychometric tests. These measurements are frequently related to factors which do not lend themselves to the exact measurements of the laboratory, and if one is to have a true measure of the value of these test results there must go along with it a feeling of these other things. This is clearly indicated by a case study at the clinic not long ago of a boy who obtained an intelligence quotient of 85. At that time he was suffering from an intense feeling of his own inadequacy to adapt himself to school and to an intolerable situation at home. Six months later, when there had been a marked change in these other things, the boy was found to have an intelligence quotient nearly 40 points above his previous one.

One should avoid making the mistake of thinking that the one way to be objective is to subject an individual to carefully controlled tests and to see only such results as having validity. The psychiatrist has a type of interest which can be just as objective, but which does not lend itself to the exact measurements of the psychometric tests. It is perfectly possible to evaluate the relationships within the family group and to be objecive about doing it and to have just the same type of reliance on facts gained through such observation as those obtained in rather carefully controlled laboratory tests.

Psychologists have been a little inclined to minimize the importance of this type of contribution, and the real value of contributions of both professional groups can come only when there is a recognition that both types of association are of importance. The field of human behavior will never be reduced to a matter of

testing, and if psychologists insist that the only way in which human behavior can become better understood is by constantly refining the test approach, or by placing increasing reliance on biochemical phenomena, it would soon become a meaningless thing because it would become isolated from the large outlook of human relationships and experiences which play such important rôles in shaping behavior.

DR. SAMUEL W. FERNBERGER: I was much impressed by one paragraph which I think exemplifies the attitude of the psychiatrist to the psychologist. The last speaker mentioned four psychologists in the Mental Health Clinic and then described their duties. At the twelfth or fifteenth duty, I lost track. It started with psychology and then on and on. That means one of two things—it means either that these four persons are not properly trained as psychologists or, if they are properly trained, the Mental Health Clinics are wasting a large proportion of time on other activities. They are not doing only psychologic work, but work which could be done by persons without psychologic training. It would be just as sensible to ask a physician or psychiatrist to be competent in chemistry, anatomy, surgery, etc., as well as in his special kind of work. The training in psychology is very specialized. It is my opinion that no psychologist is competent to do psychologic work without a Ph.D. degree and without special training in clinical psychology. It is further my opinion that the Ph.D. from the universities granting the Ph.D.'s in this country is the equivalent of an M.D.

DR. MORRIS S. VITELES: It seems necessary to repeat one or two points brought out in my paper: the first is that the tests cannot be used as a sole instrument of diagnosis. There must be added to the test results a clinical analysis of talent and defect and all of the data bearing on the individual's adjustment. The problem narrows itself down to the question of who is most competent to gather and evaluate all of the facts about the individual necessary in individual diagnosis and adjustment. I believe that the training for such work is highly specialized; that it cannot be given in the medical school as merely one unimportant medical course, but that it is given to students working in the psychologic laboratory and clinic for the doctor's degree in this field.

DR. SANDY: I am sorry that I have given Dr. Fernberger the impression that the activities were so scattered. So far as qualifications are concerned, I had one girl with a Ph.D. and just lost her. All the others conform to the requirements of a qualified psychologist and are practically ready for the Ph.D. degree.

RELATION OF THE CHILD GUIDANCE CLINIC TO THE CHILD HYGIENE MOVEMENT. DR. GERALD H. J. PEARSON.

It is recognized that many of the physical defects and disabilities of adulthood can be traced directly to inadequate attention to the physical condition during childhood or as sequelae to serious but preventable children's diseases and the attention that has been directed to the prophylaxis and early and adequate treatment for such physical conditions has resulted in a steady improvement of the nation's physical health. Unfortunately, equal attention has not been directed to the psychic health, although it is well recognized that personality maladjustments and disorders of behavior cause more intense suffering to the individual than any but the most severe physical conditions and result in a vastly greater expenditure of society's time and money.

As in the case of physical disabilities, the beginnings of such disorders lie in the years of childhood. Whether one believes in the essentially structural basis of personality, or whether one believes that it results from the interaction between the child and his environment, everyone today seems to recognize that behavior is an individual's attempt to react and to adapt himself to his environment. That is, the behavior of an adult is his attempt to obtain emotional satisfaction from his environment and is based on the manner in which he found such satisfaction by his reaction and attempt to adapt himself to his environment as a child. Of course, environment does not mean only the physical surroundings or the actual events of life, but is also the interplay of emotional attitudes between the parents,

the siblings and the child. If it is true that adolescent and adult behavior disorders and perhaps personality maladjustments result from emotional relationships during childhood, prophylactic and early treatment for such disorders during childhood should have a result as beneficial as similar measures applied to the physical health have had. Although these concepts have been recognized for centuries, it is only in recent years that practical application has been made of them, but sufficient time has elapsed to show that such measures produce worthwhile results.

Many cases could be cited to indicate that prophylactic and early treatment for personality and behavior disorders can be applied in childhood with as beneficial results as have been obtained by similar measures in physical conditions. Practically, treatment measures of this sort can be applied only through such organizations as child guidance clinics for several reasons. They are time consuming, particularly when parental attitudes have to be altered through the working out of the parents' own maladjustments. The factors that produce personality and behavior are very complex, and their study and treatment requires the coordinated efforts of psychiatrist, physician, psychologist and social worker. Such a fourfold method of approach not only ensures a thorough study of the whole situation and the most advantageous application of treatment, but prevents a lopsided point of view of the case. Of course, certain problems perhaps are handled best by one member of the group.

Child guidance clinics offer a particularly well integrated service for the treatment, both actual and prophylactic, for disorders of personality and behavior. Through treatment of the total situation, that is, of the emotional relationships between the parents and the child, the latter may be saved from carrying certain attitudes into adult life which may become evident as serious antisocial behavior. He may be freed also from continuing certain emotional relationships with his parents into his adulthood. Although such relationships are normal for certain stages of his development they become abnormal and show themselves as psychoneurotic symptoms if carried into adult life. It is recognized generally that the prolonging of these emotional relationships with the parents into adulthood is the basis for many of the psychoneuroses. It may also prevent the child from becoming psychotic later. As treatment is applied not to the behavior of the child, but to the total situation to which the child's behavior is a reaction, it helps the parents to a better adjustment to each other and to themselves, and often it is found that the child's problem is not really as important as that of the parents. Treatment of the parental attitudes not only benefits the child indirectly but also, by aiding the parents themselves to a better adjustment, benefits them directly and prevents other children in the family from becoming entangled in their difficulties, from which entanglement they would tend to develop problems of behavior and personality.

However, the treatment of problems is not the sole contribution which a child guidance clinic makes to the mental hygiene of a community. The meaning of behavior is only dimly understood as yet and the clinic because of both its organization and its clientele, is in a unique position to investigate scientifically the factors which may determine behavior and influence the personality. It can conduct researches into the prognosis of behavior and personality disorders. As so much of its treatment is purely experimental it offers an opportunity for the study of various methods and eventually may be in a position to determine what type of treatment is the most advisable to other persons and agencies working with personality and behavior disorders on a less intensive plane, and so place the treatment for all such disorders on a more scientific plane. A child guidance clinic, because of its organization, its experimental approach to all problems and its clientele, contributes to the mental hygiene of a community by an attempt at a really scientific plan of treatment of individual cases, by research into the whole problem of behavior and personality and by disseminating the results of such researches among other persons or social agencies engaged in the management of behavior problems.

Book Reviews

CHILDREN'S BEHAVIOR AND TEACHERS' ATTITUDES. By E. K. WICKMAN. Price, $2. Pp. 247. New York: The Commonwealth Fund Division of Publications, 1928.

This short monograph is one of the few oustanding contributions to the field of social psychiatry, particularly that dealing with behavior problems in children, because it lays emphasis on the concept that problems of personality and behavior in children are fixed and accentuated, if not actually produced, by the attitudes of adults toward the children and toward their behavior, certain phases of which may be normal childish characteristics. As the author points out in the introduction, personal and social attitudes are important factors in the solution of any human problem, but whereas the influence of attitudes toward physical and mental disorders affects chiefly the treatment of these diseases, attitudes toward behavior are an integral part of behavior disorders.

Behavior in the social sense is a socially evaluated and socially regularized product, and behavior problems represent conflicts between individual behavior and social requirements for behavior; the very existence of a behavior problem is designated by a social or personal attitude, for there can be no problems in behavior in the active social sense unless some one reacts to them as such. Any form of conduct in a child or adult may become a problem if it is regarded and treated as undesirable behavior by the social group in which the person lives.

In ordinary practice, the factor of attitudes is often forgotten in the behavior equation. When a parent or teacher is distressed by the behavior of the child, the usual assumption is that the difficulty is with the child. If behavior problems of children are defined in terms of bad, evil or wrong behavior, the natural causation cannot be appreciated. Fortunately, people are beginning to think more objectively about disorders of conduct and to evaluate child behavior in terms of child welfare. (Unfortunately, the welfare of the child seems not infrequently to be confused with the convenience of the adult.) From an objective point of view, the issue in the distressing behavior of a child becomes: 1. Why is the adult distressed by the child's problem? 2. Why does the child behave in a fashion that distresses the adult? Any attempt to study and treat behavior problems of children involves an analysis: (1) of the child whose behavior is distressing; (2) of the social order that declares the behavior unacceptable or unwholesome, and (3) of the interactions between them, for it is impossible to consider a child's behavior apart from the attitudes taken toward his conduct. In the study reported in this monograph, the author attempts to bring to conscious recognition how some adults — i.e., teachers — behave toward the misbehavior of children. (On account of the method, no attempt could be made except by inference to inquire into the why of this adult behavior, although, as the author points out, such an inquiry would be of great importance.)

The study itself is an attempt to inquire into the prevailing attitudes of teachers toward child behavior: (1) their habitual method of regarding child behavior with reference to the kinds of behavior which they consider undesirable or unwholesome; (2) their customary responses to those problems. Three groups of teachers were studied: one in a selected Minneapolis Public School, one in a selected Cleveland Public School and one consisting of small groups of teachers in various public and private schools and attending instructional courses in the state of New York. Although identical methods were not applied to each group, the scheme utilized was largely as follows: 1. The teachers were asked to list the behavior problems they had encountered during their teaching experience. 2. The teachers' differential attitudes toward various types of problems were obtained by three measurable units. On appropriate rating scales that permitted quantitative scoring, the teachers rendered their individual reactions to the problems themselves, to pupils in whom the problems were observed and finally to the total behavior adjustments of these

pupils. The results obtained from the first questionnaire indicate that teachers are most aware of those problems that affect the child's application to school tasks and are more sensitive to overt types of behavior and aggressive personal traits than they are to the personal problems of children. Behavior problems are observed by teachers to occur more frequently in boys than in girls.

The observations in this part of the study demonstrate the truth of the author's definition of behavior problems; i.e., that any behavior may become a problem if it is so regarded and treated by the adult to whose care and training the child is entrusted.

Chapter 5 discusses the way in which a problem child is identified as such by a teacher, and again it is found that teachers fail to interpret many problems in child behavior as symptomatic of educational, social or emotional maladjustment. Only when the behavior of a child is of a certain distressing kind and exhibited to an extreme degree is significance attached to the behavior disorder. As a general rule, teachers tended to list the various types of behavior of children in the following order of seriousness:

	More serious than	More serious than	More serious than
Immoralities, dishonesties, transgressions against authority	Violations of orderliness in classroom, faulty application to school work	Extravagant, aggressive personality and behavior traits	Withdrawing, recessive personality and behavior traits

In contrast to this, a group of mental hygienists rated the same traits as to their seriousness for the future adult adjustment of the child as follows:

	More serious than	More serious than	More serious than
Withdrawing, recessive personality and behavior traits	Dishonesties, cruelty, temper tantrums, truancy	Immoralities, violations of school work requirements, extravagant behavior traits	Transgressions against authority, violations of orderliness in class

In the eighth and ninth chapters, some of the reasons for these opposing points of view and the effect of the teacher's attitudes on the developing personality of the child are discussed. It seems that the reactions of teachers (and not only those of teachers but those of the majority of adults dealing with children) to behavior problems of children are largely determined by the direct effect which the behavior produces on the adult. The usual treatment is directed toward the undesirable behavior, which is the symptom of maladjustment, instead of toward the underlying causes that produce the maladjustment. The very attitudes taken by the teachers toward behavior disorders in children — assailing, as they do, the attacking types of behavior and indulging the habits of withdrawal and dependency — entrench, instead of remedy, the unhealthy modes of response.

In the tenth chapter, the author draws attention to one of the most important concepts of social psychiatry; namely, that teachers' attitudes are themselves problems in behavior, and that they have developed out of the life experiences of the individual teachers. Any attempt to instruct teachers in the meaning and purpose of the behavior of their pupils must take this concept into consideration, for only as the emotional and social adjustments of the teachers' personal problems become stabilized, can they encounter unemotionally and treat rationally the exhibition of undesirable behavior in children.

Dr. Wickman shows how teachers' attitudes intensify and crystallize personality and behavior problems present in children when they enter school. It lies beyond his purpose to show how these problems arise in the relationships between the child and the adults with whom he comes in contact and the attitudes of the latter toward him during the preschool period. Such a study is needed urgently, and it is to be hoped that the Commonwealth Fund will sponsor such a study as a supplement to the present one. The reviewer, perhaps, has written an unnecessarily long review of such a small book, but it is felt that the attention of educators, jurists, pediatricians, psychiatrists and, particularly, physicians in general should

be drawn to this monograph, not so much for the factual data it contains, as for the philosophy which underlies Dr. Wickman's approach to the whole question of behavior and personality; for the great majority of persons who actually have to deal with these problems seem often ignorant of well recognized and proved pertinent facts.

THE PHYSIOLOGY OF LOVE. GEORGE M. KATSAINOS. Price, $4. Pp. 326. Boston: Privately printed, 1929.

Every neuropsychiatrist has had young people come to him in great distress after they have read pseudoscientific books depicting the terrible consequences of sexual irregularities. "The Physiology of Love" by Dr. Katsainos belongs to this class of books. One can easily see that if this book falls into the hands of naive young persons or even uninformed adults it will cause a great deal of mental anguish and drive the reader to Dr. Katsainos' office for last minute aid against the "irremediable injury" he has incurred.

What clear ideas one can disentagle from the highly bombastic circumstantial style of expression he uses seem to be mostly wrong: that children would never experience a sexual urge if they were not specifically taught by others; that erection in young people when they come to maturity leaves "absolutely no impression on the imagination"; that in human beings the sexual feeling is not instinctive; that before the ages of 23 in men and 20 in women sexual intercourse is harmful, physically and intellectually, rendering the individual generally inferior; that "dreams do not occur during sleep, no, but at the moment of waking . . .," the dream being "an act of wakefulness and not of sleep. . . . In other words, no such thing as a dream exists"; that the only difference between animal and man is the faculty of speech; that all patients who masturbate "have without exception been initiated into vice by a teacher of their own age"; that "those who masturbate, whether male or female, are impotent . . . when called upon to function normally they show themselves to be absolutely and irremedially incompetent"; that the cause of all sexual perversions is early masturbation; that "only one part of the male body . . . interests the woman . . . without this adjunct man has for woman the same value that a mechanical doll has . . . his virility, his companionship, his value depend on that organ and on no other part of his body"; that to place a female patient "nude on all fours and ask her to walk in this attitude" is a procedure which "we physicians often have to do professionally"; that certain perversions lower the individual to the lowest human stage forever; that if syphilis and gonorrhea should disappear, the institution of marriage would cease, because it is kept intact only by the fear of these diseases; that homosexuality is caused only by excessive intercourse with women; and so on.

The author, who writes that he has treated persons with venereal diseases for thirty years, is evidently unable to examine his patients, especially female ones, without considerable affect. He tells, for example, how a young girl, "scarlet with embarrassment," came to him for an examination which he undertook "with no less embarrassment." He then goes on for one and one-half pages to describe in flowery, lyrical language the beautiful details of this "virginal pudendum." He takes a strong stand against Freud, especially on account of the book "Psychopathia sexualis." This criticism of Freud is somewhat invalidated by the fact that he does not seem to realize that this book was written not by Freud, but by Krafft-Ebing! For the lay reader, the book is misleading, especially in its frequent use of the word "irremediable" to characterize the evil consequences of sexual irregularities and its references to insane asylums as the fate of some of his patients.

If a book of this sort deserves mention in a scientific journal, it can be only to point out the great harm it can do in the hands of the nonmedical readers, to whom it is evidently addressed. The author complains that a previous publication of his was barred by the Massachusetts State Library and the Boston Library "not on account of its contents, far from that, but of its title 'Syphilis.'" But, judging from the contents of the book under review, that does not seem to have been an instance when Boston censorship should be blamed.

UEBER UMBAU UND ABBAU DER SPRACHE BEI GEISTESSTÖRUNG. F. G. STOCKERT. Price, 7.80 marks. Pp. 82. Berlin: S. Karger, 1929.

Stockert has produced a monograph of great interest and importance. His approach to the problem of speech through the utterances of mental patients has resulted in new concepts in the psychology of speech. The monograph is readable, and should be available to all neurologists and psychologists. To those interested in the problem of aphasia the monograph is replete with new avenues of approach to this extremely difficult subject.

Speech is presented fundamentally as an expression of the individual in his adaptation to his environment, as a means of influencing others, as a purposeful act, and as a method of symbolization. It is important to look on speech not as a mere isolated act, but as a highly complicated physical and psychologic act whereby the individual reacts to the environment and evokes reactions in the latter simultaneously. From the pathologic standpoint Stockert divides the disturbances of speech as observed in mental cases into disturbances of motor expression, and disturbances of symbolization. With regard to the former he states that there is a definite and characteristic rhythm of speech for every person, characterized by certain dynamics, pitch, etc. This is the physiognomy of speech which can be modulated and varied in proportion to the perceptive powers of the individual. The lesser the perceptive power, the greater is the monotony of speech. Speech, moreover, in its musical basis, has a certain tempo, pitch and inflection. All of these vary in the individual, and also in mental cases. The speech of the neurotic person varies in rhythm, tempo, and pitch, and expresses in a clear way the insecurity of the life of such an individual. The speech of anxiety neurosis is characterized by long pauses which are later overcompensated by torrents of words.

Speech, moreover, has a definite melody, manifest in the conscious accent of the sentences. That this factor is important is shown by the different meanings which may be placed on sentences of the same content, but differently accented. This melody of speech is disturbed in many conditions. In diseases with cortical pathologic changes it is evidenced by monotony, as in the dementias, such as those of paretic, senile and epileptic types. The speech of paresis is characterized by a similarity of content day in and day out—a constant repetition of the same ideas. Epileptic persons have a monotonous, arrhythmic speech characterized by its inconsequentiality, its failure ever to arrive at a point. The same is true of senile dementia. In all these forms one sees the impossibility of a unified grammatical expression of the thought process. The sentence which is started is lost or is repeated because it never was properly formed, a function that is impossible in the general slowing of thought. The monotony noted in these conditions, however, is lost in excitement; speech then becomes a dialog with a true melody. Pick has pointed out that melody is the most durable element in speech, and is the last to disappear in aphasias.

The symbolic function, as is well known, is the most highly developed element in the speech of man; it is this element that is lacking in the speech of animals. It is disturbed in mental diseases, as in the paralogias of schizophrenia. The fundamental difficulty consists in a difficulty of understanding. Either only isolated facts are grasped and woven into a whole, or the various elements are not seen in their correct relation to one another and are woven into a false system, as in the case of schizophrenia.

DIE NEUROPATHOLOGISCHEN SYNDROME. Zugleich Differentialdiagnostik der Nervenkrankheiten. By DR. M. KROLL, Professor, Director Der Nervenklinik der Weissrussichen Staatsuniversität Minsk. Price, 45 and 48 marks. Pp. 532. Berlin: Julius Springer, 1929.

This volume does not lend itself to extensive abstracting. It is one of the few works on neurology which have appeared from Russia since the war, and in that respect alone it is of great interest. In a volume of 532 pages, the author discusses practically the whole subject of the new neuropathologic syndromes,

with the differential diagnosis of organic nervous diseases. It is, in effect, a discussion of the newer points of view of the last two decades. It is divided into twenty-six chapters. The classifications are in themselves interesting. There is first a syndrome of disturbances of movement. Practically all new works on neurology pay great attention to the advances in this field, and deservedly so, for our knowledge of movements, particularly those arising as the result of striatal diseases, has increased greatly.

The second subject is the syndrome of sensory disturbances; the third, the reflex disturbances; the fourth, electrical irritability; the fifth, the cerebrospinal fluid; the sixth, the cerebellar syndromes; the seventh, aphasia; the eighth, the frontal lobes, and the ninth, the temporal lobes. The tenth and following chapters deal, in order, with the syndromes of the different parts of the brain, and there are special chapters on diseases of the extrapyramidal system; the epilepsies; symptoms resulting from increase of intracranial pressure; spinal pressure; circulatory disturbances; neurosyphilis; meningitides; epidemic encephalitis; endocrine disturbances, and angiotrophoneuroses. Finally, there is a ten-page discussion of the syndrome of the neuroses.

The book is well illustrated, mostly with original matter. The subject is well discussed, but it is notable that there are few American references. This probably is because the author has had the advantage only of continental literature; practically the only references to American work are through publications in continental journals.

L'APPAREIL VESTIBULAIRE DANS LES TUMEURS CÉRÉBRALES. By ISAAC ALFANDARY. Price, 25 francs. Pp. 213. Paris: Gaston Doin, 1928.

This monograph, from the service of J. A. Barré, Professor of Neurology at the University of Strasbourg, reviews the anatomy, physiology and semiology of the vestibular apparatus in its first part and its disorders in increased intracranial pressure, tumors of the cerebellopontile angle, the brain stem, the cerebellum, the fourth ventricle, the cerebral peduncles and hemispheres in the second. There is a good bibliography.

Alfandary first recalls the observations of Souques and of Babinski, made in 1904, that increased intracranial pressure affects the eighth nerve in the same way as the second, and that the former may be affected when intracranial pressure rises, irrespective of the location of a brain tumor. He also reemphasizes the now well established fact that cerebellar disorder does not produce nystagmus. Alfandary regards the question of cerebral representation of vestibular impulses as still unsettled, thus omitting the considerable body of evidence now at hand establishing this. The chapter devoted to increased intracranial pressure emphasizes the high percentage of vestibular symptoms (77 per cent) as compared with the frequency of choked disk (90 per cent). Of these, vertigo is common. Nystagmus, according to Alfandary, never occurs due only to increased intracranial pressure. He has not found any vestibular syndrome characteristic of increased intracranial pressure, but regards the progression of various symptoms which do occur as of importance in diagnosis, though not of localizing value. The process of blocking the transmission of impulses centralward by a tumor results in absence of reaction to peripheral stimulation (caloric, for example) and irritation at the point of blocking with subjective abnormal sensations (tinnitus, for example). This is a condition like that of anaesthesia dolorosa.

There are good case reports and frequent allusions to the literature. The book is well written. For those interested in either the vestibular apparatus or brain tumors, it is well worth reading.

THE ARCHIVES OF NEUROLOGY AND PSYCHIATRY is published by the American Medical Association to stimulate research in the field of diseases and disorders of the nervous system, and to disseminate knowledge in this department of medicine.

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The ARCHIVES OF NEUROLOGY AND PSYCHIATRY is published monthly. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75, including postage. Single copies, 85 cents, postpaid.

Checks, etc., should be made payable to the AMERICAN MEDICAL ASSOCIATION.

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